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NOCTURNAL SYMPTOMS.

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IT is not the first time that I have dealt with the subject of nocturnal symptoms. More than once in a long clinical career I have made the bold attempt to approach and enter the vast labyrinth of the nocturnal symptoms (Engel, 1922, 1926); but every time I have had to realize that the most thorough knowledge of up-to-date physiology and pathology did not prove to be a reliable Ariadne's thread and I have failed. I could not, however, help keeping my attention focused on the topic. For the last two decades I have persistently studied the literature of the world. I found a number of articles dealing with one symptom or another, but I searched in vain for an informative paper summing up the whole field; I was not able to discover a single one. So I will undertake a new trial myself. Maybe the widening of the field of patho-physiology gives me a chance.

In my former papers I have set out as my main aim the finding of answers to two questions: (i) Can we get some contribution to the analysis of a symptom from its nocturnal appearance? (ii) Can we get significant help in the making of a diagnostic conclusion from the occurrence of a symptom during the night?

That a symptom coming at night may have its characteristics and may eventually offer some differential diagnostic evidence is well known. A simple mention of such symptoms promptly calls for the association with a probable disease. Nocturnal pains in the upper part of the abdomen suggest the idea of duodenal ulcer; colicky pains, a gall-bladder attack; generalized convulsions, epilepsy; nocturnal dyspnœa, cardiac or bronchial asthma. The symptoms may appear early in the night, in the "small hours" of the night or later, nearly at dawn. And the realm of nocturnal symptoms is not restricted. We hear from a large number of our patients that some of their symptoms occur at night, only at night, mostly at night or at night also.

By the term "night" I do not mean here the cosmic night, with its characteristic metereological changes—darkness, changes of temperature, of barometric pressure, humidity, electric conductivity, content of cosmic rays *et cetera*. Much as one would be inclined to accept the possibility that metereological changes of such extent might influence somatic processes, there is little conclusive scientific evidence to be found for it (Frey, 1949; Castellani, 1931). So far the best known is the influence of darkness provoking symptoms such as night-blindness, terror of the dark, deterioration of the gait of tabetics *et cetera*. But this small group takes a subordinate place only in comparison with the much larger group of real night symptoms.

By "night" here I intend the night of the human being: the time when it is retiring to a quiet rest, is staying in bed, is abstaining from any food, and, what is by far the most important, is asleep. Night in this sense expands to about one-third of the 24-hour day; hence it is natural that symptoms occur at night corresponding to the laws of probability. Naturally these are not true nocturnal symptoms. Real nocturnal symptoms are the ones which in all patients occur for a long period in their lives or even for their whole lives exclusively at night or mostly so. This type is represented by nocturnal epilepsy; some patients have their fits for years and for decades exclusively at night ("nightfitters", Griffiths and Fox, 1938).

There are several factors acting in the genesis of nocturnal symptoms. Sometimes one of them is sufficient to produce a symptom; in other cases only the synergic action of several can cause an effect or integrate them to a symptom. These factors are of a different importance and it is our task to rank them, to evaluate the merit of each one and to determine when and how they cooperate. Let us deal with them in a proper order.

The first one is the bedroom: the room itself, the air, the smell and the dust in it, the bed, the pillows, the mattresses (feather, kapok), all of which are able to provide allergens by which anaphylactic symptoms can be provoked coming on later in the course of the night. A short stay in the room at daytime may not deliver antigens enough, but a prolonged stay in bed does.

A further important factor is lying in bed. The relaxation of muscles in the prolonged recumbent position of sleep releases their protecting fixing mechanism. This is the reason for sudden pains which awaken the patient suffering from rheumatic fever or from rheumatoid arthritis, from spondylarthrosis, from tuberculous inflammation of the hip joint *et cetera*, which do not let him fall asleep again. If pains arise or grow stronger somewhere in the muscles of the body during the night, the first presumption has always to be that they are of arthritic origin; but pains due to fibrositis of the muscles may also increase during the night. Sometimes the "growing pains" of children become very intense during the night. "In sacroiliac arthritis the pains are generally much more severe at night because in a recumbent position the normal lumbar lordosis is obliterated and therefore more strain is thrown on the joint" (Mercer, 1945). It is well known that the pains irradiating from the shoulder along the whole upper limb (brachialgia, the scalenous syndrome) may become much more severe at night. Sometimes severe pains in the arms may start at night with accompanying and, later on, stabilized vasomotor disturbances in patients who sleep with their arms extended upwards, in which position both the subclavian artery and the brachial plexus are under pressure between the clavicle and the first rib (*nyctalgies paresthésiques*—Leriche, 1948; Wright, 1945; Froment and Garde, 1947). Quite a slight compression of this kind may cause the not infrequent complaint of numbness in one or both upper limbs, most distinctly in the hands. It is felt on the patient's awakening in the morning and generally passes in a couple of minutes. It occurs mostly in women and never appears in the same form in the lower limbs.

It is very important that in the prolonged recumbent position of sleep compression symptoms caused by tumours may appear or may be intensified. I have a vivid memory of several such patients.

I remember one patient with a large cerebellar tumour (confirmed at autopsy) who was startled almost every night by a sudden excruciating headache, with other consistent symptoms of increased intracranial pressure, by a very slow pulse and by intense vomiting. He hardly complained about headache in the daytime.

Another patient with a tumour of the spinal cord did not dare to lie down to sleep for months without having taken a strong analgesic, as the prolonged recumbent position provoked the most atrocious girdle pains.

There are cases described in the literature in which patients with a tumour of the brain had to be awakened at night and sat upright because of the impending signs of respiratory paralysis. But it is important to keep in mind that the same may be observed in cases of encephalitis. Sometimes sciatic pains caused by a protruded disk become much more severe at night in about 25% of cases (Love, 1947). Because of the importance of the matter I shall give two quotations from the most recent literature.

Adson and Svien (1949) make the following statement:

> If the patient [with a tumour of the brain] is ambulatory headache occurs daily in the early morning hours and frequently awakens him at about 4 a.m.

Adson (1949) has the following remarks to make:

> The ache invariably awakens the patient [with a tumour of the spinal cord] from four to six hours after he has retired. It becomes often so severe as to compel him to walk the floor or to sleep in a sitting position.

The rest pain in *arteriosclerosis obliterans* of the lower limbs is important. It is most commonly noted at night and consists of a dull to a moderately severe persisting aching pain, which may interfere with sleep and often causes the patient to sit up and rub his feet for hours at a time. The same can be observed in Buerger's disease (Allen, Barker and Hines, 1946).

The changing of the circulation, especially of the venous blood, is of great importance in the horizontal position of sleep. In this posture the head is situated rather low and so the return of the venous blood from it is somewhat hindered. The pressure in the intracranial veins rises from the zero of the perpendicular position to about six to eight millimetres of mercury (Best and Taylor, 1945). On the other hand, the return of the venous blood from the lower limbs is promoted in this position. This is the reason why œdema of the lower limbs decreases or vanishes in course of the night, particularly of course if it is caused by venous congestion. (On the contrary, the face, especially the eyelids, becomes sometimes swollen in such cases, because of the increased venous pressure in this area.) The resorbed œdema fluid finds its way into the circulation and later into the urine. This is the cause of nycturia (or nocturia), increased production of urine during the night. Koranyi (1929), undoubtedly one of the best authorities in this field, has adopted the term "postural polyuria" to indicate the postural origin. I will deal circumstantially with this exceedingly significant symptom later.

When the subject is in the recumbent position the blood pressure is somewhat lower, the heart action slower. The blood volume is somewhat increased. Both halves of the diaphragm are more elevated than in an upright position. This may be an important factor contributing to several symptoms (oppression, shortness of breath), especially in cases in which the two halves of the diaphragm have been already pushed upwards otherwise (ascites, meteorismus, pregnancy).

A further factor is the uniform warmth of the bed. This is obviously a condition which may initiate the onset or exacerbation of cutaneous symptoms. Pruritus often torments its victims only at night—the general essential pruritus as well as that caused by jaundice, uræmia and pediculosis, and anal and vaginal pruritus. *Oxyuris vermicularis* comes out of the anus mostly at night and contributes to the itching in the perineal region. I shall never forget the veritably terrific nights I had to spend because of frenzied itchings and scratchings when I acquired scabies after the capture of Budapest by the Russians; during the day I did not have the slightest sensation of any distress. Molesworth (1944) believes that the nocturnal exacerbation of symptoms is due to increased activity of the parasite. The prolonged warmth of the bed is certainly a contributing factor in nightly erections and emissions and in the frequent micturition of the prostatics.

I would mention here that serious symptoms may appear in connexion with the sexual act generally performed at night (dyspnœa, *angina pectoris*, apoplexy).

Sleep.

The most relevant of all factors surely is sleep. It plays an absolutely fundamental role in the ætiology of nocturnal symptoms. Many a "nocturnal symptom" may appear during sleep in daytime too (sleep symptoms). Because of its dominant significance I shall discuss sleep here *in extenso*. As it would be irrelevant for the scope of this paper, I will refrain from discussing the theories of sleep, "the eternal mystery", from dealing with its phylogenetic evolution and from treating the many conceptions of sleep centres and their localizations. On the other hand, I will deal in detail with the alterations in the functions of the whole organism and its single organs. There are many; but I will stress of course only those which may play the role of provocative agents (Best and Taylor, 1945; Trömner, 1912; Pieron, 1913; Laache, 1913; Salmon, 1930; Kleitman, 1939; Evans, 1947).

One of the most characteristic features of life is the alternation of activity with rest. In animals on the highest scale of evolution it appears in the alternation of the waking state with sleep. The biological aim of sleep is obvious and can be established without being teleologically oriented. It is to guarantee the most complete rest possible

for the whole organism and for each of its parts with the intention of building fresh potential energy for further activity. The mechanism of sleep, however, is still completely obscure. Its problem has remained unsolved so far, despite all the reasoning of the greatest intellects and the best scholars for thousands of years.

Sleep is surely not a purely passive condition, it is not simply an inactivity of the central nervous system induced by exhaustion. Sleep is a specific, well-integrated active condition of the brain, in which the activity of certain of its parts is almost completely at a standstill, that of others is partially switched off, and finally there are parts of which the activity remains practically intact. The ability to sleep is not linked with the cortex; dogs or pigeons with their cortex removed fall asleep at regular intervals, but the sleep periods are shorter than in normal animals. During sleep the will, consciousness, orientation for time and space, and reactivity to moderately strong stimuli from the outside world are at a standstill. The mind is in complete sleep. In the cortex some cellular activity continues, and this is the biological background of dreams. But even the functions mentioned before do not cease completely. Man in sleep does react to stimuli to a certain extent: on feeling cold the sleeper draws up his blanket; some people awake at a fixed time. In exceptional circumstances the brain continues mental work and the sleeper awakes in the morning with a complete solution of the problem (the morning call "Eureka!", or Kekulé's famous discovery of the benzene formula—Japp, 1898).

In the electroencephalogram of sleep reduced or δ waves appear; experts see in this finding evidence of the reduction of cortical activity.

Let us treat in succession the manifestations of the changed functions both of the whole organism and of its organs.

The picture of the sleeper is that of a person in a passive, immobile posture, with closed eyes, relaxed muscles, quiet, regular, slightly slowed respiration.

In sleep the functions of most excretory glands are lowered. The secretion of tears is already diminishing at the approach of sleep. The drowsy person rubs his eyes; he tries to overcome the odd feeling of dryness in his eyes by mechanical stimulation of the lachrymal secretion. The German legend says: "The Sandman is coming." Because of diminished salivary secretion, the lips, the tongue and the throat of the sleeper with open mouth are dried. Nasal secretion decreases; it is well known that nasal discharge becomes less profuse in sleep even in a case of severe acute coryza, and it may even stop completely. The production of sputum also diminishes. In the absence of any cough reflex the sputum steadily accumulates in the bronchi. This is the reason for sudden coughing attacks which interrupt the sleep or burst out in the morning, generally with the expectoration of copious sputum. Opinions about the secretion of gastric juice were divergent until recent times. On the basis of the most recent reliable investigations we know now that the gastric secretion continues unchanged all night during sleep in normal persons as well as in those with gastric or duodenal ulcer (Levin *et alii*, 1948*a*). The volume and concentration of acid in the fasting nocturnal gastric secretion are usually higher in patients with duodenal ulcer than in normal persons (Levin *et alii*, 1948*b*; Saundweiss *et alii*, 1946; Woegtlin, 1947). The secretion of bile is diminished, mostly because of the absence of food intake.

The secretion of urine is strongly diminished. The ratio of nocturnal urine to that of the daytime is about 1:2. This is the reason why a normal person hardly passes any urine at night. The solid content of nocturnal urine is not so much reduced as its water content, hence the concentration of the urine is rather high, approaching the specific gravity of 1·030. The mechanism of the diminished secretion of urine has been well clarified by the most recent search of Sirota *et alii* (1950). They found as follows:

> During the four hour period from 12 midnight to 4 a.m. corresponding to the period of deepest sleep there is a slight but significant fall in glomerural filtration; not in renal plasma flow. The decrease in urine flow at night is almost wholly attributable to increased tubular reabsorption of water.

To my mind the increased tubular reabsorption points towards an increased pituitrin secretion. In cases of *diabetes insipidus*, in which the pituitrin secretion is lowered or abolished, the quantity of urine does not diminish at night. The sleep is greatly disturbed, as the patient persistently has either to drink or to pass urine during the night.

Of all the secretory glands, only the function of the sweat glands is increased during the night. Generally the whole body surface of the sleeper is moist, because of abundant sweating. According to Hartridge (Best and Taylor, 1945), the quantity of liquid lost in sleep by sweating is nearly equal to that lost during a corresponding period of strenuous muscular exercise. The increase in sweat production is on one hand caused by the continuous uniform warmth of the bed and on the other hand by central nervous influence.

We hardly know anything about the function of the glands of internal secretion during sleep. Some of these seem to have their functions diminished, though not to the same extent as the glands of external secretion. However, as there are no conclusive data in the literature, suppositions are rather hypothetical.

The peristaltic function of the gastro-intestinal tract continues in sleep. The stomach empties itself within the first half of the period of sleep. The contents of the intestines are moved along probably with a reduction of the intestinal mobility. As people in the morning generally pass a stolid stool, resorption from the bowels must have been normal. The passing of intestinal gases is absent during sleep; passing of flatus starts only on awakening. In the case of abnormal gas production as a consequence of fermentation, the accumulation of gases may cause distension with gas to a considerable extent. In such a case the already elevated halves of the diaphragm are pushed upwards even higher and may cause serious distress.

As stimuli for opening the *papilla Vateri* and evacuating the gall-bladder are not present in sleep, the papilla remains closed. Hence stasis is developed in the gall-bladder, which may become the starting point of severe gall-bladder colic appearing characteristically at night-time.

The rate of the heart action drops more in sleep than in a waking subject in a simple recumbent position. The minute volume is not reduced below the basal level (Best and Taylor, 1945). The blood pressure is lowered; the fall may extend to 10 to 30 millimetres of mercury. The fall is larger than in simple recumbency. The fall of blood pressure in sleep was suspected and demonstrated a long time ago; recent investigations have proved these previous results entirely correct. The lowest level of blood pressure is reached at about the fourth hour of sleep. This level then is maintained until a short time before awakening, when the pressure begins to rise and reaches the normal level on awakening.

The pathologically increased blood pressure may also drop during sleep. But the behaviour of the fall is not the same in different kinds of hypertension. In cases of essential hypertension the blood pressure falls to a larger extent than in normal persons. The fall may amount to 15 to 100 millimetres of mercury and so the pressure may sink to the region of the normal level. In contrast, the blood pressure of malignant hypertension remains more or less unchanged in sleep; in cases of uræmia it may even increase. The behaviour of the blood pressure in sleep corresponds, therefore, to that in the "Avertin" test. However, the drop in the diastolic blood pressure is not so pronounced in sleep as that of the systolic blood pressure (Best and Taylor, 1945).

The cause of the fall in blood pressure is dilatation of the arterioles in the periphery (proved by the plethysmo-

graph), which is produced by an alteration in the functions of the vasomotor centres.

Many interesting alterations are to be found in the blood during sleep apart from those already mentioned. The most important is that the carbonic acid content of the serum is increased, the chemical reaction is shifted to the acid side, the pH usually is rather less than 7·4. These signs of pronounced acidosis indicate lessened excitability of the respiratory centre. In the presence of its normal regulatory function such changes could not have been brought about. The fact that respiration of the Cheyne-Stokes type can be observed very often during sleep may be regarded as a further sign of the same alteration. I would stress the great importance of this alteration; it surely plays a considerable contributing role in the genesis of nocturnal symptoms.

One of the most thrilling findings belonging to this chapter is that microfilariæ of *Wuchereria bancrofti* are found in much greater abundance in the peripheral circulation at night than in the daytime. Towards evening they begin to appear in gradually increasing numbers until about midnight, when an enormous quantity can be found in every drop of blood: 300 to 600 filariæ. After midnight their number decreases gradually. This periodicity can in time be reversed if the individual sleeps by day and is up at night. The appearance of the filariæ is therefore linked with sleep—it is a real sleep phenomenon. In Loa Loa infection the microfilariæ are more abundant in the circulating blood during the daytime than at night. The periodicity of filariæ has never been satisfactorily explained (Strong, 1942).

The basal metabolic rate is distinctly diminished in sleep. It is about 10% lower than in recumbency alone (Best and Taylor, 1945). The diminution reaches its maximum at the time of the deepest sleep. It is caused by the absence of movements and by the decrease in the tone of the striated muscles in sleep. Obviously parallel with this the body temperature decreases slowly in the course of the night, and the lowest level is reached at about dawn.

In some cases the shape of the temperature curve of febrile conditions may exhibit some difference from the normal; in other cases it resembles the normal curve. The falling of the temperature in some highly febrile diseases may be extremely rapid in the early morning hours and may therefore be of considerable significance (the tendency to collapse at dawn of patients suffering from pneumonia).

The tone of the striated muscles is diminished in sleep—the muscles are relaxed almost everywhere, the limbs are loosened. In some muscles, however, a good deal of the tone may remain—for example, in the respiratory muscles. Horses sleep in the upright position, birds in the sitting position. The rigidity of patients suffering from Parkinson's disease is less stiff, contractures are somewhat relaxed. All kinds of trembling, shaking, choreatic, athetotic, myoclonic and spasmodic movements are diminished, or may even stop completely in sleep. The tone of the diaphragm also decreases; the form of respiration therefore tends towards the pectoral type. The relaxation of the diaphragm augments (or increases) the elevation caused by the recumbent position. Because of this elevation of the diaphragm the vital capacity is diminished. Lowering of the tone of the soft palate contributes to snoring. Relaxation of the muscles is the reason why tendon reflexes are generally less brisk in sleep. A number of authors have reported the diminution, even the abolition, of abdominal and cremaster reflexes, a group of others an up-going toe in deep sleep; but these observations have not been confirmed by other investigators.

Closing of the eyes for sleep is hardly a simple consequence of muscle relaxation, because in this case the lower lid would have to fall; there must be an active contraction of the *orbicularis oculi*. This seems to be confirmed by the finding of some resistance on trying to move the upper lid upwards. When the subject is awake the shutting of the eyes is followed by dilatation of the pupils. In sleep the pupils are constricted. This cannot be produced in a simple way by the relaxation of the dilator pupillæ—the sphincter has to contract actively too.

The sleeper generally does not react to a slight touch, to lesser changes in the outside temperature, to moderate stimuli of light and sound. He reacts to a stronger light with blinking, and a still stronger light interrupts his sleep. The perception of pain is diminished. Severe pain makes the sleeper groan, very severe pain awakens him. Therefore the conclusion is that if a pain interrupts sleep it is surely not a slight one. Severe pain of course interferes with falling asleep. The body weight decreases in the course of sleep, as the output of carbonic acid by respiration and loss of water by insensible perspiration and in the expired air are going on steadily.

A critical interpretation of the symptoms described leads to the final conclusion, that in sleep the tone of the sympathetic system is diminished and that of the parasympathetic increased. "Sleep is essentially a parasympathetic integration" (Foulton, 1943). Increase of the tone of the sympathetic system is the characteristic of the state of emergency (Canon, 1914), of the state of alarm (Selye, 1937). Sleep is just its opposite: "Sleep is a deficiency in preparedness for animal activity" (Hess, 1932). Consequently it can be established and maintained only by the opposite mechanism. The increased tone of the parasympathetic system is the cause of the slowing down of heart action, of dilatation of the peripheral vessels, of constriction of the pupils and of the tendency to erections. The supposed (Conderelli, 1932), but not definitely proved, deterioration of the coronary circulation in sleep because of constriction of the coronary arteries fits well into this scheme; it is well known that the innervation of the coronary arteries is the opposite to that of the systemic arteries.

Once more I would emphasize the depression of the respiratory centres in sleep, because this alteration plays an absolutely dominant role in the provocation of important nocturnal symptoms.

Nocturnal Symptoms.

After this biological introduction I will now turn to the description of nocturnal symptoms.

The Onset of Labour.

First, a word about labour. Formerly I believed that more than 50% of labours started at night. There seemed to be some data to support this, but they have not been confirmed. In the words of Busby (1949): "The general consensus of opinion today is that both onset of labour and delivery are equally divided around the clock." Earlier observations that hæmorrhage caused by *placenta prævia* occurred more often at night than in the daytime have not been confirmed either.

Insomnia.

An important nocturnal condition is insomnia. I need not deal here with the different varieties or its causes, because there are many excellent monographs on the subject. I will just mention it because it is the cause of a series of symptoms—palpitation, vasomotor troubles, headaches *et cetera*. On the other hand, many a symptom of an organic disease (tachycardia, dyspnœa, frequent micturition) produces insomnia. A most pronounced vicious circle may develop in both ways.

Psychosomatic Symptoms.

The short periods before falling asleep and following awakening are the times of psychosomatic symptoms. The neurasthenic never falls asleep as quickly as is desired. In the darkness of the bedroom, tossing about, he soon starts to ponder. His attention is not diverted by the categoric demands of daily work, so he begins to look inwards. Very soon the otherwise imperceptible manifestations of his organic functions reach the threshold of apperception and organ consciousness begins, especially that of the heart. The most varied odd sensations rush on (palpitation, extrasystoles, constriction in the heart, throbbing of arteries, sensation of alternating heat and

cold, numbness of the limbs *et cetera*)—a legion of them, which are described eloquently in the characteristic, bombastic, affected phraseology of the neurasthenics. The same mechanism produces the psychosomatic symptoms after awakening at dawn. In the morning depressive symptoms are prevalent and may quickly turn into desperation with suicidal intentions. The early morning is indeed the time when most suicides are committed. "It is about five in the morning, you know, that suicides are most common" (Conan Doyle). However, reliable scientific statistics are so far not available.

It is most important, however, from the diagnostic point of view, that the times of occurrence of psychosomatic symptoms are, as already mentioned, the period before falling asleep in the evening and that after awakening in the morning, and not the period of sleep itself. During sleep the psychodynamic symptoms pause. Neurasthenic trigeminal neuralgia—a not infrequent and very often misunderstood syndrome—pauses during sleep, as do a legion of other psychosomatic pains; but the pain of real trigeminal neuralgia mercilessly awakens the slumberer from his best sleep. Cardiac pain in aortic regurgitation often occurs during sleep, awakening the patient in terror (McBryde, 1947). Nervous tachycardia stops during sleep; the tachycardia of heart failure, of fever or of exophthalmic goitre is merely slowed down a little. The nervous patient who in daytime passes urine every half hour has a quiet sleep; but the person with catarrh of the bladder is awakened from his best sleep by the stimulus of urgent micturition. The patient suffering from nervous diarrhœa never gets up at night to empty the rectum (Alvarez, 1943). If the patient is awakened from his sleep by a running nose, the condition is most likely, almost certainly, allergic (Vaughan, 1948). I could quote a number of similar examples, and I feel, therefore, that it is very important to notice and to keep the following dictum almost as a rule: that symptoms interrupting sleep are signs of organic conditions and are not functional. Exceptions of course cannot be ruled out. It is sometimes not easy to establish whether the patient has been awakened by a pain or only felt it after awakening—for example, whether he felt an anginal pain on turning to the side or on sitting up to pass urine.

Epilepsy.

Old observations have already reported the frequency of epileptic seizures during the night. From our point of view real nocturnal epilepsy is more important, because the seizures appear only at night-time—that is, in sleep. The patient feels a severe headache in the morning, a peculiar fatigue, signs of bites on the tongue, and finds the wet bed—all of which demonstrate the event of the night. That there certainly is some connexion with sleep may be revealed by the fact that such patients have their seizures in daytime only when asleep. Oppenheim (1926) introduced the term "hypnogen epilepsy". As the mechanism of both epilepsy and sleep is completely obscure, the connexion betwen the two naturally cannot be clarified. It is important from a differential diagnostic standpoint that hysterical fits never occur in sleep; the occurrence of general convulsions in sleep therefore establishes the diagnosis of epilepsy.

Sleep Palsy.

"Sleep palsy" is the term for a kind of paralysis of the radial nerve (rarely of the ulnar nerve), which occurs during sleep as consequence of a long-lasting compression in a certain position. It mostly befalls alcohol addicts, as only the narcotic effect of a large quantity of alcohol can produce the depth of sleep for hours which is necessary for such a long-lasting compression of the nerves by a hard object without awakening the patient ("Saturday night paralysis"). It is likely that the greater vulnerability of the nerves of alcohol addicts is also a contributing factor.

Sleep Paralysis.

"Sleep paralysis" is the name for a very rare disease, in which flaccid paralysis of the nature of paraplegia or quadriplegia is developed mostly at about midnight with an odd feeling of stiffness. The tendon reflexes and electric irritability are lost. It generally lasts only a few minutes and never longer than till about noon the next day. The patient is fully conscious and painfully aware of his helpless condition. The pathogenesis is completely obscure.

Nocturnal Enuresis.

Nocturnal bed-wetting is interesting mostly for the pædiatrician. In the literature it is emphasized that a number of children wet themselves if they sleep in the daytime (Hobhouse, 1932). The conditioning factor is sleep, not night. If bed-wetting occurs in adults who did not suffer from it in childhood, it may be the first sign of a serious organic condition (tabes, disseminated sclerosis, chronic meningomyelitis).

Syphilis.

In the symptomatology of syphilis described in older text-books the excruciating nocturnal pains played an outstanding role (*dolores osteocopi nocturni*). They mostly assumed the form of a very intense headache coming at about midnight, but sometimes lasting the whole night. Other patients felt pain of the same character in the lower extremities or in the region of the lower part of the back. Not infrequently similar pain occurred in the daytime when the patient was asleep. But an expert in this field, Schlesinger (1925), terms the pains in arthrolues real nyctalgias; he observed many cases in which the pain did not appear during sleep in the daytime. The most relevant factor in the mechanism of the pain is obviously the circulatory change caused by both sleep and posture, with acute swelling of the diseased tissues (mostly gummata of the skull or other superficial bones) and consequent irritation of the sensory nerves. It is remarkable and striking for me, since I have been dealing with the subject for so long, to find in the newer text-books on syphilis only a few short remarks about these once so dreaded pains. Kampmeyer (1946) and Stokes *et alii* (1944) are of the opinion that the nightly occurrence has been over-emphasized in the literature. I think that the reason for this is the changed picture of syphilis. Tertiary skin manifestations have disappeared almost completely nowadays; tertiary bone alterations have lost much intensity as a consequence of modern treatment.

It has, of course, to be borne in mind that not every nocturnal headache is of syphilitic origin. Severe headache at night can be caused by a tumour of the brain (mechanism already mentioned), by uræmia; even the pain due to glaucoma, to iridocyclitis and to caries of the teeth may become much stronger at night. Real migraine starts mostly after the awakening in the morning; sometimes, however, the patient awakens with a well-developed attack. Very characteristic is the morning headache of patients with hypertension.

Symptoms of Diseases of the Circulatory System.

Let us follow now the symptoms caused by diseases of the circulatory system. The best known are those of congestive heart failure. The bed, the recumbent position of sleep, are adverse to the patient with uncompensated heart disease. He cannot remain in a recumbent posture, not even on high cushions. As soon as he lies down he feels distressed, oppressed, he does not get enough air, he begins to cough and he has to sit up very soon or quit the bed. He cannot find sleep in a recumbent position. Everyone remembers one or many heart patients dozing with a livid face in an armchair, or rather with the head sunk on the clasped hands on the table. The heavy and noisy breathing is slowed down, the expiration is prolonged. From time to time he starts, stares wearily around, snatches for breath for minutes, grows exhausted and drops back tormented into an unquiet sleep again. Patients with uncompensated heart failures are sometimes unable to lie on the left side because of distress and palpitation. Conversely, patients with an immensely enlarged heart are

sometimes unable to lie on the right side, because in this posture the heart is jammed in the narrow space between the sternum and the spine.

It is of paramount importance that the scheme of urine secretion of the patient with uncompensated heart disease is altered; he secretes more urine during the night than during the day (nycturia). Congestion of the kidneys due to heart failure delays the excretion of water for many hours during the day, and so it is eliminated only at night. The excretion of water is delayed as much in patients suffering from uncompensated kidney disease. The reason is the asthenuria (Koranyi, 1929), loss of the ability to produce a more concentrated urine than that having a specific gravity of 1·011 to 1·012. The same type of delayed urine excretion may be observed in patients with ascites because of cirrhosis of the liver. The contribution of the posture and the role of mobilization of the œdema fluid in producing nycturia have already been dealt with.

Nycturia is a very important symptom; it is often the first sign of cardiac decompensation (precœdema) and can remain the only sign for a long time. In every case of heart failure we have to check the quantity of nocturnal urine systematically and with care; it may disclose very important evidence. If a disappearance of œdema occurs during the night without distinct nycturia, then the œdema is almost certainly due not to heart failure, but to local conditions (manifest or latent phlebectasia, flat-foot).

Patients with progressive arteriosclerosis, with syphilitic aortitis, but especially with long-standing hypertension, not infrequently awaken from their first sleep with sudden air hunger. In some cases the blood pressure suddenly rises very high (vasomotor crisis—Pal), but mostly it sinks, sometimes conspicuously. The attack may be light —slight dyspnœa with some coughing or only coughing and uneasiness lasting for some seconds or minutes. After sitting up and coughing for a minute or two the patient may feel all right, and being relieved may drop to sleep again. There are many different forms of transition from this light attack to the most severe and tormenting paroxysm of dyspnœa called cardiac asthma. The patient may be tormented for long hours until he is relieved, may suddenly succumb after a while, or may after some hours be drowned in the frothy, sanguinolent fluid of pulmonary œdema. These different syndromes are described and detailed well in text-books, and form a gratifying subject for the sometimes masterly description of human suffering. (White, 1944; Levine, 1943; Scherf and Boyd, 1948; Fishberg, 1944.) The paroxysms may occur only rarely, at intervals of weeks and months; but they may practically every night attack patients who try to continue their work during the day, until they have to give up fighting and ask for admission to hospital.

What is the reason for the real cardiac asthma occurring exclusively at night or, rather, in sleep? As a matter of fact, it grips its victims in daytime too if they are asleep. It has no connexion with the recumbent position or with the mobilization of the fluid of œdema, as has been supposed by some investigators. More than one text-book quotes cases of patients who are confined to bed because of some other ailment and have their attacks nevertheless only when asleep. Why should sleep, the best friend of mankind, turn against him, plague and kill him? Cardiac asthma is the most important and most interesting nocturnal symptom; as a matter of fact, it is the very syndrome that has aroused and sustained my interest in the topic. In the end the cause of cardiac asthma is failure of the left side of the heart. The right ventricle pumps the blood into the lungs, where it is dammed up—hence the pulmonary œdema. An efficient right ventricle is a *sine qua non*. With the advent of pronounced venous stasis the attacks usually become less intensive and less frequent or disappear, presumably because of diminution in the predominance of the right ventricle (White). But the salient point is, why does failure of the left side of the heart develop in sleep, when the patient is resting quietly, when he is not carrying on any mental or somatic labour, when he has no excitement—that is, when he has complete psychic rest? In a word the attack comes when the heart is spared most. The mechanism of cardiac asthma is far from being fully clarified, though thorough studies have well approached the problem. Every text-book of heart diseases deals with it in a more or less detailed form. A large number of papers of a high standard have appeared on the subject in the last years; as the best I nominate that of Herzog (1947). The pathogenesis is complex; each factor mentioned in the introduction is cooperating. The primary and obviously the crucial factor without doubt is the lessened irritability of the respiratory centre in sleep, with consequent disturbance of the physical regulation of the respiration; in a word, the syndrome originates in the nervous system, hence the beneficial, often indeed miraculous, effect of a quick morphine injection. I myself would stress the deterioration of the coronary circulation as a consequence of the increased vagus tone which plays a role in the constriction of the bronchial muscles too (Fishberg, 1944). The elevation of the diaphragm and its relaxation with the reduction of the vital capacity may also have a not negligible influence in the attack. The elective effect of the peculiarly integrated mechanism on the left side of the heart and on the permeability of the capillaries of the lungs makes this syndrome one of the most appealing in the whole of human pathology.

It can be assumed that anatomical alterations in the arteries of the respiratory centres and in the coronary vessels, mostly as a consequence of long-lasting hypertonia and arteriosclerosis, are also necessary; cardiac asthma occurs only in relatively advanced age and never in young people. Young people suffering from mitral stenosis with distinct pulmonary congestion never develop real cardiac asthma.

Symptoms of Diseases of the Respiratory System.

A characteristic nocturnal symptom is bronchial asthma. Many of the attacks of most patients occur at night (Colle, 1939). This is so in the early stages, as well as later on at the stage of persistent asthmatic catarrh; but there are patients whose attacks occur exclusively during sleep, whether at night or in the daytime. Our hospital in Budapest had wards where asthmatics had been collected. During the day the wards were silent, but at night there always could be heard the noisy breathing and coughing of patients from every corner of the ward. There were nights when the resident physician was called to a dozen of them to give injections. In the mechanism of this nocturnal occurrence of bronchial asthma we have to take into consideration every factor mentioned before: blocking of the nose in some patients as the starting point of the provocative reflex, constriction of the bronchial muscles because of the increased vagus tone in sleep, and particularly the effect of allergens.

I have already mentioned that coughing is generally diminished or even stopped completely during sleep as a consequence of absence of the cough reflex. In contrast to that, we hear from some patients with laryngeal or tracheal catarrh that they cough much more convulsively at night than in daytime. The attacks may imitate those of pertussis. It is obvious that in such cases the irritation of dried sputum on the tracheal mucosa provokes the attack, and this may be the mechanism of the pseudocroup (spasmodic croup) attack of children during the night too. However, the attacks of real pertussis as well may occur more frequently during the night.

Pulmonary hæmorrhage is not infrequent at night. Each of us has heard of one patient or another who has been awakened in the course of the night by severe coughing and has perceived only in the morning that he has been spitting blood. But intense or severe hæmorrhage also can occur at night. Recurrence of an already stopped pulmonary hæmorrhage at night is obviously caused by the absence of the care which such patients take to avoid coughing during the day.

Diseases of the Digestive Tract.

There are two nocturnal symptoms especially to be mentioned from the range of diseases of the digestive tract: the nocturnal pain of duodenal ulcer and gall-

bladder colic. Many patients with duodenal ulcer are awakened at about 1 or 2 o'clock by intense (or severe) pain in the epigastrium. The pain is of the character of the hunger pain, as the patient generally feels it before lunch or before dinner. The pain is generally more intense in the case of a patient who has had meat for dinner. In a large number of duodenal ulcer cases we hear that the pain is very intense in the daytime and never occurs at night; but conversely it is not infrequent for the pain to appear exclusively at night and in a very excruciating form. The pain starts at about or after midnight, because the peristalsis of the stomach in sleep is somewhat slower and so the stomach becomes empty at that time. I have already dealt with the continuance of the gastric secretion in sleep.

Persistent epigastric nocturnal pain is characteristic of duodenal ulcer. Naegeli (1936), one of the most outstanding Continental clinicians, was of the opinion that persistent nocturnal pain in the epigastrium was more characteristic of duodenal ulcer than hunger pain. In spite of that, it cannot be regarded as crucial. Exceptionally, similar pain can occur in cases of gastric ulcer, of severe gastritis, and especially of gall-bladder disease.

That gall-bladder colic occurs often at night has been well known for ages. It is important that the milder cramp especially comes at night both in the early and in the later stages of the disease. However, severe attacks calling for an urgent injection of morphine are also not infrequent during the night. The physiological background of the stasis of bile in the gall-bladder because of sleep has already been dealt with.

Other Symptoms.

In some text-books I have found some mention of the frequency of renal colic at night.

An important sign of hypertrophy of the prostate is frequent micturition during the night. This may occur at a comparatively early stage, at which the patient has no complaints during the day. The cause is obviously acute hyperæmia of the prostate because of the recumbent posture, warmth of the bed and sleep. Sudden higher grades of such alterations may produce the most dreaded acute retention of urine—a not infrequent tragic syndrome of the night. In the later stage of the disease with residual urine, frequent micturition after midnight is more or less obligatory and may seriously interfere with quiet sleep.

A typical nocturnal symptom is the attack of gout (Hartridge). The patient is awakened mostly after midnight by excruciating pain in the toe. Sydenham (1683) describes how he was aroused at 2 a.m. by the agonizing pain in his toe. The articulation appears swollen, reddened and tender, and the patient is febrile. Generally he drops into an unquiet sleep only at about dawn. In daytime he does not feel so bad, the pain is well tolerated; however, it recurs systematically during each of the following nights at about the same time in a slowly decreasing intensity until after a period of a week it ceases completely. The reason for the nocturnal occurrence is not clear. Early investigators revealed that the uric acid excretion is diminished a good deal at night. It cannot be ruled out that this may be a synergetic contributing factor.

In persons suffering from gout, and also in others with different metabolic diseases (uræmia, intoxications), in persons with varicosities, and in alcohol addicts, the well-known spasmodic contractions of the muscles of the calves may appear in the night (cramps—Moss and Hermann, 1948). Frequent appearance, somewhat longer duration or greater intensity of the cramps causes a serious disturbance of sleep. Details of the mechanism of these symptoms are completely unknown.

Nocturnal sweating of patients with tuberculosis of the lungs is notorious. It is well known that it may become so profuse that the patient has to change his completely wet shirt two or three times. I mentioned in the introduction that of all glands with excretory functions, only the sweat glands function more intensely at night. A somewhat warmer blanket and a drop in the increased temperature cause the excessive sweating. It is important, however, that such sweating may occur without any rise in temperature as an initial symptom. A toxic factor as a contributing cause cannot be ruled out in such cases.

Hæmoglobinuria.

Nocturnal hæmoglobinuria (Marchiafava-Micheli syndrome) is very interesting from a theoretical point of view. It is a rare syndrome, mostly not severe at all, and has no serious consequences whatever. Most of the investigators are of the opinion that it may be connected with acidosis of sleep (Wintrobe, 1946; Whitby and Britton, 1942). The red cells of the affected subjects are susceptible to a potent lysin which is present in normal serum and which acts particularly at pH 7·0 to 7·2. The increased blood destruction occurs only during sleep, whether it be in the day or at night, and is not related to posture or to fluid or food intake.

Death.

Finally, I just mention here the most important, the final, supreme symptom, death. There are few reliable data about the hours of death, but it is evident from them that death is not a nocturnal symptom. "There are more deaths during the hours of day than at night" (Bogar, 1941).

Conclusion.

I have arrived at the end of my study. I am well aware that there are many more nocturnal symptoms in my branch of internal medicine, as well as in other lines of medicine, but on this occasion I cannot deal with them.

And if after all that I return to the two questions asked in the introduction, I have to answer them in the following way. To establish the nocturnal appearance of a symptom may indeed offer real help in the differential diagnosis, and as a matter of fact is sometimes decisive. The answer to the second question is, however, in the negative. The pathogenesis of the majority of symptoms of diseases is so far so obscure that their occurrence at night does not contribute towards clarification. The problem remains unsolved and is therefore present. There is an urgent need in this field for the most intensive work of many investigators—physiologists, pathologists and clinicians equally. I have tried to arouse their interest and to give them some directions in this modest study; let me hope that my endeavour has not been completely futile.

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RECURRENT PTERYGIUM.

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IT is well established that pterygia occur to a much greater extent in tropical and subtropical climates than in colder climates. Pterygium is a common ocular lesion in Queensland and attains a fairly high incidence in the north, especially in the north-west, where to the effect of temperature is added irritation by dust and glare.

Some idea as to the frequency of pterygia in Queensland may be gathered from the fact that from 350 to 400 pterygia are removed each year at the Brisbane General Hospital, where the research work recorded in this paper was carried out.

Unfortunately, irrespective of the particular surgical technique used in removing the pterygium, the recurrence rate is also high in hot climates.

Various modifications of the transplant operation are employed by practically every Queensland ophthalmic surgeon, and it is well known that transplanting the head of the pterygium does not prevent recurrence of the lesion.

Until recently it was common practice to perform repeated transplant operations on recurrent pterygia. I have seen one removed for the tenth time and have myself removed several pterygia which had been transplanted on three or four previous occasions. After several such operations the condition almost invariably becomes progressively worse. The pterygium recurs in a short time and becomes tough, fibrous, highly vascular, and firmly adherent to the cornea and sclera, so that there is actually some danger of penetrating the eyeball when the surgeon is attempting thorough removal of the pterygial tissue. All these facts were brought out at a discussion on recurrent pterygia at the last Australasian Medical Congress (British Medical Association) (following a paper on the subject by K. B. Redmond, 1950), where it was agreed that the recurrent pterygium frequently becomes a serious ophthalmological problem.

In view of the known high incidence of recurrent pterygia in Queensland, I commenced an investigation of the subject last year at the Brisbane General Hospital. A series of 240 patients with pterygium were carefully examined pre-operatively with the aid of the slit lamp, again one week after operation, and finally three to nine months after operation. The head of each pterygium was removed and sections were examined. I also recorded the history, including the family history, occupation and residence in various parts of Queensland, and finally the appearance of the cornea and limbus immediately after operation.

This last observation is important, since a large percentage of pterygia do not dissect off in a well-defined plane, as is often implied in accounts of the subject. Frequently the surgeon is compelled to dissect off numerous tags on the cornea in order to make sure of removing all pterygial tissue. It is sometimes difficult to decide how far to pursue this object, since over-zealous attempts may create new tags and ridges, and not uncommonly one finds, at the limbus, that the knife is cutting into the superficial layers of the sclera, incidentally with bleeding from the anterior ciliary vessels. The alternative is deliberately to shave off a thickness of the cornea beneath the pterygium, which is a satisfactory procedure provided one does not go too deep. The point is that if an appreciable amount of tissue is left on the cornea near the limbus a recurrence becomes much more probable, and for that reason it is aimed to remove all pterygial tissue and yet leave, if possible, a smooth cornea. It is relevant to remark here that, contrary to what is often stated in text-books (Duke-Elder, 1939), the majority of operations for removal of pterygia do not leave any obvious scar on the cornea, which is in fact cosmetically transparent unless too much corneal tissue has been removed, and unless, of course, the pterygium recurs.

The initial operations in this series were shared in approximately equal numbers by Dr. Brian Wilson (registrar of the eye department, Brisbane General Hospital) and myself. The same technique was used, except that Dr. Wilson left a much narrower "bare area" than I did. Incidentally, our recurrence rates were almost identical. The exact technique is described in Figures I to III. In a number of cases, after the head had been removed from the cornea, I completely excised the subconjunctival connective tissue in the manner described by D'Ombrain (1948) before continuing with the transplanting. Although the actual recurrence rate was much the same in these cases, the recurrent pterygia were flatter, less vascular and cosmetically better, and I have now included this step as a routine measure.

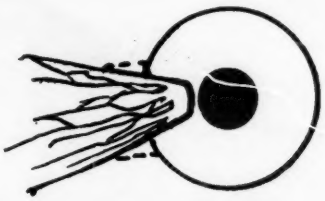

FIG 1

Incisions are made in the conjunctiva above and below the pterygium; the head of the pterygium is then dissected off the cornea.

Incidence of Recurrence.

It is first of all necessary to define recurrence. At the discussion mentioned previously, K. B. Redmond referred to revascularization of the cornea (even by a few vascular strands) as a recurrence, and T. Boyd Law (1950) defined a recurrence as a regrowth of vessels on the cornea or persistence of the red appearance of the eye.

Assessed by these standards, fully 50% of the pterygia removed in this series recurred, since in this percentage of cases revascularization of the cornea to over 1·5 millimetres from the limbus occurred at intervals of three to nine months after the operation. In the majority of these cases, however, the eye was cosmetically satisfactory and the appearance of the scar ranged from near transparency to varying degrees of translucency when examined by focal illumination. Time alone will tell the ultimate condition of these patients, whose state will be referred to in future not as recurrence, but as "revascularization of the corneal scar".

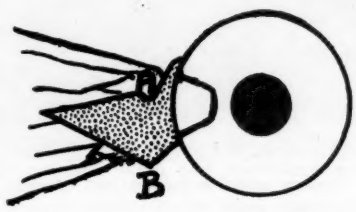

FIG 2

A triangular area of conjunctiva is excised, including the lower half of the pterygium.

True recurrences—that is, recurrences of well-vascularized conjunctival tissue obvious to both the patient and the surgeon—took place in 20% of the series. However, some of these true recurrences were cosmetically better than the original pterygium, and no further surgical treatment is proposed for the present in these cases. If persistence of the red appearance of the eye is included as a criterion of recurrence, then the recurrence rate is even higher, since a number of eyes which had no revascularization of the cornea were obviously red over the inner half of the exposed bulbar conjunctiva.

The results are set out in Table I.

Factors Involved in Recurrence.

Let us now examine the various features of the pterygia in order to ascertain if there are any features correlated to recurrence.

Age.

The ages of the patients ranged from fifteen to sixty-nine years. Several patients aged over sixty years had a large, fleshy, heavily vascularized pterygium encroaching to or beyond the centre of the cornea. There were only a few patients aged under twenty years. This is partly because there is a comparatively low incidence in this age group (although pterygia occur in children as young as nine to ten years of age), and partly because most surgeons are of the opinion that there is a higher incidence of recurrence in these young patients, and on that account the operation is deferred if possible to a later date. The results of the present series fully confirm this opinion and show a progressively lower recurrence rate with each decade of life. The results are set out in Table II.

TABLE I.

Results of Pterygium Transplanting in 240 Cases.

Final Condition of Eye Three to Nine Months After Operation.	Percentage of Cases.
No recurrence of vessels on the cornea and mostly a quiet eye	50
Revascularization of the cornea for 1·5 to 2·5 millimetres from the limbus, but cosmetically satisfactory	30
Obvious recurrence of vascularized conjunctival tissue—that is, recurrent pterygium	20

TABLE II.

Relation Between Age and Incidence of Recurrence.

Age in Years.	Percentage of Patients with Recurrence of the Pterygium (Well Vascularized Conjunctival Tissue).	Percentage of Patients with "Revascularization of Corneal Scar".	Total with Recurrence of of Vessels on Cornea. (Percentage.)
Under 20	20	60	80
20 to 29	40	30	70
30 to 39	20	33	53
40 to 49	20	20	40
50 to 59	15	15	30
60 and over	—	30	30

Family History.

In every case an attempt was made to obtain a family history of pterygium. In a high percentage of cases the patients declared that they were not aware of pterygia occurring in any of their family. However, a number stated that several members of their family had pterygia; for example, one woman noticed pterygia in both her parents and in two of her four sisters, which is a rather high family incidence. Again, I observed a well-defined pterygium in the right eye of one patient and also in the right eye of both his son and his daughter. This, of course, may be a coincidence, and must be considered in conjunction with environment. It is of interest, however, that in the case of both father and daughter the pterygium recurred after operation.

Briefly, there appears to be an hereditary factor in pterygium, but the available evidence was not sufficient to enable this to be accepted as a fact.

Rate of Growth of Pterygium.

The rate of growth was considered from two angles. The first was the patient's observations as regards the rate of growth in recent months. In this respect no correlation was found between rate of growth and incidence of recurrence. Secondly, in every case the distance of the head of the pterygium from the limbus was measured and compared with the period of time which had elapsed since the patient first became aware of the lesion. Again there was no relation between apparent rate of growth and recurrence.

FIG 3

The head of the pterygium is transplanted downwards, a bare area being left (stippled area).

Sex, Occupation, and Environment.

Of the patients, 60% were males, the large majority of whom were outdoor workers in a dusty atmosphere, engaged, for example, in farming, sheep or cattle station work, motor-truck driving, and so on. There is therefore a definite relation between occupation and incidence of pterygia; but there was not an appreciably higher incidence of recurrence amongst the outdoor workers as compared with indoor workers. Of the series, 48% came from the north or the west of Queensland, which confirms the opinion that there is a relatively high incidence of pterygium in these areas. Since the large majority of these patients are now living in or near Brisbane, no idea could be obtained as to the effect of the northern and western environments on the recurrence rate.

FIG 4

Diagrammatic sketch of pterygium showing anastomosing vessels, grey edge with irregular projections, corneal island opacities and well-defined pigment cap.

Slit Lamp Appearance of the Pterygia.

Details of the pterygium are easily observed with the slit lamp, retro-illumination showing up clearly the extent of the corneal opacities and blood vessels.

The pterygium is translucent, with a mesh of anastomosing horizontally disposed deep and superficial vessels. A number of pterygia showed small subepithelial cysts resembling bubbles and also patches of hyaline degeneration. At the apex is a grey edge of varying size and transparency, and usually with a number of grey, irregular projections, beyond which, in most of the pterygia, were small, irregularly shaped, corneal, opaque areas. In a number of pterygia the grey edge appeared relatively very wide and continuous with a coalesced mass of corneal "island" opacities. A not uncommon occurrence was the presence of a partial or well-defined pigment cap lying outside the grey edge. These features are shown diagrammatically in Figure IV.

On the available evidence there was no relation between the degree of multiplication of the opacities at the apex and tendency towards rapid growth, as stated by Berliner (1943), or at any rate there was no relation to recurrence tendency. However, the results did agree with Berliner's other statement that extension of the vessels into this zone indicates a tendency towards rapid growth, if this term is taken to mean tendency towards recurrence. Those pterygia with vessels extending well up to the edge showed a higher recurrence rate than the average. However, this does not mean that these pterygia will necessarily recur.

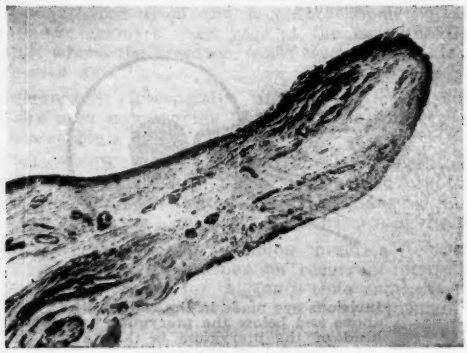

FIGURE V.

Showing the usual epithelium, loose subconjunctival connective tissue and vessels.

FIGURE VI.

Showing hyperplasia and dyskeratosis of the epithelium with "balloonization" of cells.

Vascularization of the Pterygium.

As regards vascularization of the pterygium, the outstanding feature in this series was the high percentage of pterygia which were well vascularized, and therein lies the key to the high recurrence rate in Queensland. T. Boyd Law (1950) found that 100 out of 135 consecutive pterygia at Lismore were vascular, and he also found a comparatively high incidence of recurrence. In this series 90% of the pterygia were vascular. The results confirmed the often-stated opinion that thick, fleshy, well-vascularized pterygia have a high tendency to recur. However, once again it should be pointed out that these pterygia do not invariably recur.

Pigment Cap.

Finally, the pterygia with a well-developed pigment cap showed a lower incidence of recurrence than the average. Out of 30 patients with such pterygia, seven showed "revascularization of the corneal scar" after operation; but a large number of these patients would need to be examined before the results could be accepted as significant.

Histopathology.

As was stated previously, the head of every pterygium in the series was excised at operation and sections were examined microscopically. A careful study of the sections did not bring to light any histological features associated with a tendency towards recurrence.

FIGURE VII.
Showing œdema, free blood, heavy vascularization, and chronic inflammatory changes (thick fleshy pterygium).

Examination of the slides revealed the usual histological features of pterygia. The pterygium consists of conjunctival epithelium with subepithelial connective tissue containing a fair amount of elastic fibres and patches of hyaline degeneration which stain a homogeneous pink colour. The epithelium in some sections showed metaplasia

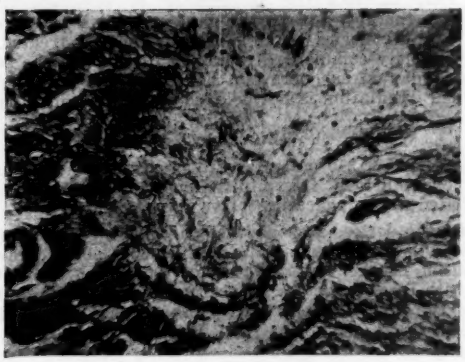

FIGURE VIII.
Note the swollen elastic fibres (elastosis).

or hyperplasia, and some showed tubular downgrowths, while others showed "balloonization" of the cells. A few sections showed "elastosis" (basophilic alteration of swollen elastic fibres). A number of these features are shown in Figures V to VIII. As was stated above, the histopathology of the pterygium does not supply any additional guide to the probability of recurrence.

Operative Technique.

The following points were observed as regards operative technique. (*a*) There is a high tendency towards recurrence if an appreciable amount of pterygial tissue is left on the cornea, especially at the limbus. (*b*) While it appears advisable to leave some bare area at operation (see Figure III), the width of this area shows no relation to the rate of recurrence. For example, although I left approximately 5·0 millimetres of bare area and Dr. Wilson left only 2·0 millimetres, our recurrence rates were the same. I have made this observation quite apart from the present series. (*c*) Excision of the subconjunctival connective tissue together with transplanting does not prevent recurrences in Queensland, but gives a better cosmetic appearance and a flatter recurrent pterygium. (*d*) Carbolization of the subconjunctival connective tissue without transplantation and without excision of this tissue, as described by Kamel (1946), has given disappointing results in Queensland.

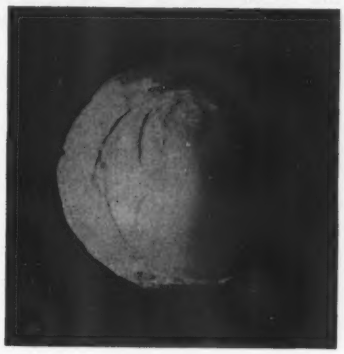

FIGURE IX.
Plaster cast.

Post-Operative Care.

Post-operatively, avoidance of dust, glare and similar irritation did not prevent recurrences.

FIGURE X.
Lead "contact lens".

With regard to seasons, there was just as high a recurrence rate in winter as in summer. Finally, in none of the series was chronic conjunctivitis present.

Treatment of Recurrent Pterygia.

The only methods which are successful in the treatment of recurrent pterygia in Queensland are the following: (i) the use of grafts, either of skin or of buccal mucous membrane; (ii) radiation therapy, especially with contact X rays. Of these methods, the use of skin grafts is usually successful, but they are unsightly, and when the graft is removed about six to twelve months later the pterygium sometimes recurs. Mucous membrane grafts give much better results; but the simplest and most successful method is to use contact X-ray therapy after removal of the recurrent pterygium.

Contact X-ray therapy has been used recently in Brisbane at the Queensland Radium Institute with very good results. The technical details of the contact X-ray

therapy and the dosage, which were worked out by Dr. A. G. S. Cooper, Director of the Queensland Radium Institute, are as follows: (i) Philips contact X-ray machine; (ii) 45 kilovolts, two milliamperes; (iii) 20 millimetres focal skin distance; (iv) one millimetre of aluminium (half-value layer one millimetre aluminium); (v) 2000*r* at surface of conjunctiva (total dose), divided into four or five doses over ten days.

I treated 18 patients with recurrent pterygium by this method and was able to improve on the already excellent results by the introduction of a "lead contact lens" in place of the strip of lead which was being used by the

FIGURE XI.
Thick recurrent pterygium.

Institute to protect the cornea. In each case an impression was taken of the eye with the recurrent pterygium and from this first a plaster cast (Figure IX) and finally a "lead contact lens" one millimetre in thickness was made

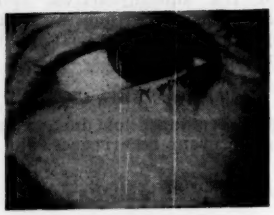

FIGURE XII.
Same eye four months after removal of the recurrent pterygium, followed by a course of contact X-ray therapy. Note the pale area giving a good cosmetic appearance.

(see Figure X). This lead contact lens was introduced into the conjunctival sac (after the instillation of several lots of "Decicain" drops, 1%) at each radiation treatment. While the patient is fixing a target with the other eye, the lead contact lens is adjusted so as to leave the scleral site of the pterygium and a narrow strip of cornea at the limbus exposed to the rays. The tube of the machine rests on the lead contact lens and the rays are directed obliquely towards the pterygial area on the sclera—that is, away from the cornea. It was found better to "flare out" the opening in the lead contact lens to almost 180°, as this gave better exposure of the top and bottom edges of that part of the limbus previously covered by the neck of the pterygium. Apart from a variable amount of soreness of the eye following each dose of irradiation, no complications occurred. The operative technique employed to remove the recurrent pterygium, prior to radiation therapy, was similar to that shown in Figures I to III, except that in all cases the subconjunctival tissue was excised and a wide bare area was left (at least five millimetres). Half of the cases of recurrent pterygia came from the present series, while the other nine cases were recurrences following operation by experienced ophthalmic surgeons whose technique was known to me. In 14 of the cases the pterygia had recurred once and in four they had recurred twice.

In every case the contact X-ray therapy was followed by the creation of a well-defined pale area corresponding to the bare area left at operation (see Figure XII). This pallor of the bulbar conjunctiva gives an excellent cosmetic result which apparently persists. For example, I recently examined a patient who had undergone irradiation therapy over one year previously, and the eye still showed the pale area medial to the cornea with a good cosmetic appearance. In every one of the 18 cases of recurrent pterygia treated as described above the cornea showed no revascularization three to five months after operation, and in most cases there was no obvious scarring of the cornea. One case is of special interest.

FIGURE XIII.
Large, flat, tendinous type of pterygium.

This pterygium was a thick, fibrous pterygium with a grey, gelatinous edge extending beyond the centre of the cornea, and vascularized with many fine vessels extending onto the cornea for about three millimetres. It was discovered that this pterygium had been removed for the second time eight months previously and that the patient had been given contact X rays. On investigation, however, I found that the patient had not reported to the Radium Institute until two weeks after the operation for the removal of the pterygium. I removed the pterygium for the third time, and contact X-ray therapy was commenced on the second day after operation, the usual course being given. A feature of the operation was the relative absence of bleeding, due, no doubt, to the effect of the previous radiation therapy on the blood vessels. Three months later, the eye was perfectly quiet, there was no vascularization and no obvious corneal scar, while a wide pale area gave an excellent cosmetic result.

This case stresses the importance of commencing the radiation therapy within the first five days after operation. In the series of 18 patients whom I treated, the large majority commenced radiation therapy on the second or third day after operation, but results are satisfactory provided irradiation is not delayed beyond the fifth day.

Summary.

1. A series of 240 patients with pterygium in Queensland were examined to determine what factors, if any, were associated with recurrence after operation.

2. Of the series, 20% showed partial or complete recurrence of the pterygium, and a further 30% showed revascularization of the corneal scar for 1·5 to 2·5 millimetres, but with good cosmetic appearance. Thus in half the series some revascularization of the cornea occurred.

3. The histopathology of the heads of the pterygia revealed no factors associated with recurrence.

4. Dusty outdoor work and residence in northern and western Queensland are both associated with a high incidence of pterygium. There is also some evidence of an hereditary factor in pterygium.

5. The following factors are associated with recurrence. (*a*) There is a progressively lower recurrence rate with higher age. (*b*) Vascularity, especially if the vessels extend well up to the advancing edge of the pterygium, is associated with a high incidence of recurrence. (*c*) Adequate removal of all pterygial tissue at operation lowers the recurrence rate. Other helpful factors are excision of the subconjunctival tissue, a bare area being left, and possibly transplantation. (*d*) Pterygia with a well-defined pigment cap appear to have a lower incidence of recurrence.

6. A series of 18 patients with recurrent pterygium were treated with contact X rays, a lead contact lens being used to protect the cornea. In every case, when the patient was examined three to five months after operation, the eye was quiet, there was no revascularization of the cornea and no obvious corneal scar, and there was an excellent cosmetic appearance due to a well-defined pale area over the original scleral site of the pterygium.

Acknowledgements.

I wish to thank Dr. B. G. Wilson for his enthusiastic cooperation and his assistance with the initial operations. I also wish to thank Dr. J. V. Duhig and the staff of the pathology department at the Brisbane General Hospital, especially Dr. J. H. Little and Miss H. Junner, for their assistance with the sections. I am also indebted to Mr. G. Thomson for the excellent photographs, and to Mr. J. Elliott, who made the lead "contact lenses".

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MACROCYTIC ANÆMIA OF PREGNANCY.

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THE impression from student days and from reading routine text-books in obstetrics is that macrocytic anæmia of pregnancy is a rare disease dependent for its development upon malnutrition and poverty. In fact, if one was anxious to study the disease it was said to be necessary to go to India, where, as Eden and Holland (1937) state, "it is very common and where it accounts for many maternal deaths".

The fact that it is not so uncommon and that it does not depend for its development entirely upon malnutrition and poverty is something gradually realized by us over the past three years. In the obstetrical practice of one of us, proven macrocytic anæmia has been as common as a toxæmia of pregnancy severe enough to require surgical induction of labour.

Most important in this series of cases is the fact that we have been able to diagnose it in patients in whom the hæmoglobin level has almost always not dropped below 70% of 14 grammes *per centum*. This contrasts with the usual picture of the condition, as exemplified in the recent series reported by Ungley and Thompson (1950) and Patel and Kocher (1950), in which all the patients had a hæmoglobin level of between 20% and 40%. This earlier detection of the condition is an important means of reducing maternal ill-health and infant stillbirth or ill health as well as perhaps of preventing the onset of premature labour.

These patients are sifted out of all those who present themselves for routine antenatal care as follows. Patients are usually examined first when about eight to nine weeks pregnant and at this stage are sent to a hæmatologist for determination of blood group, Rh factor and hæmoglobin level. All patients at their initial visit are put onto a daily dose of five grains of ferrous sulphate. When the hæmoglobin finding is returned the dosage of iron is left unchanged if the hæmoglobin level is over 80%. If it is less than 80% the dosage is increased to either 10 or 15 grains of ferrous sulphate per day, according to the degree to which the hæmoglobin level falls short of 80%. For the patients with this low hæmoglobin level in whom the response to iron is not satisfactory, a full blood examination is then obtained. Generally speaking, patients with macrocytic anæmia belong to this group.

The evidence of non-response to iron is generally a persistence of symptoms that can best be described as neurasthenic. The patients are languid and irritable and are finding their pregnancy a burden. We do not consider that the normally healthy person should find her pregnancy a great burden.

What we have described is the routine by which we now sift out these patients, but in the first patient of this series the discovery of the macrocytic type of anæmia was quite accidental. Mrs. V.O. at her first visit said: "I have always been anæmic and I have always been taking iron." On the basis of this statement a complete blood examination was carried out at once, and this revealed that the hæmoglobin level was 80%. At every subsequent visit this patient had a fresh complaint, and in an endeavour to relieve symptoms she was given tonics, barbiturates and amphetamine, all without relief. At the seventh month of the pregnancy, by which time her visits were being anticipated with a certain amount of dread, another full blood count was ordered. Although the hæmoglobin level was 90%, the blood film showed a considerable degree of macrocytosis.

It was the combination of a high hæmoglobin level with macrocytosis and symptoms of neurasthenia which led us to be on the lookout for this condition, and now in any patients with neurasthenic symptoms and no apparent physical or environmental circumstances to explain the symptoms, the possibility of macrocytosis is always considered. The statement that this patient made about "always taking iron" is perhaps significant, because five of the patients with the condition have made the same remark to us.

The most gratifying aspect of these patients is the dramatic response to appropriate therapy. All the patients say "I have never felt so well in my life", and this improvement is a source of pleasure not only to the patient, but to the medical attendant and, not by any means the least important, to the husband.

Husbands report an improvement in the patients' feeling of well-being, and quite a considerable improvement in temper, as their symptoms of languor and weariness are relieved. Another way in which it is beneficial is typified by the statement of one patient who, a week after the institution of therapy, said: "You know, doctor, I feel so much better, and a week after I started on the pills I got up and got my husband's breakfast for the first time since I have been pregnant." At this stage the patient was seven months pregnant.

The social importance of health as a means of maintaining harmony in a home needs no emphasis. We think that this is a very important reason for being on the lookout for this condition, because all such patients are much happier and, we are sure, much easier to live with when they get the appropriate treatment.

The beneficial effects are not appreciated by the husband only. Because these patients feel so much better, they handle their new babies with more assurance. It is the tired, weary woman who develops physical and mental problems in the course of rearing her family. In the well woman these problems are very much less likely to arise.

This series of cases is too small for us to attempt any assessment of the bearing of the condition upon the nature of the labour, the health of the baby, or the influence of treatment upon lactation, although we can say that in every case labour was normal, the child appeared quite healthy, and lactation was adequate. One patient was discovered to have this condition in her second pregnancy. In the first pregnancy labour had been premature—

TABLE I.

Summary of Findings in Cases of Macrocytosis.

Patient.	Months Before Term.	Hæmoglobin Value. (Grammes *per Centum*.)		Duration of Treatment in Weeks.	Lymphocytes per Cubic Millimetre.	Mean Corpuscular Volume. (Cubic μ.)	Mean Corpuscular Hæmoglobin Concentration. (*Per Centum*.)	Diet.
		Before Treatment.	After Treatment.					
Pen.	3	8·0	11·2	2	405	—	—	Good.
A.	4	10·5	11·5	12	1010	—	—	Good.
Vo.	1	12·6	14·7	3	995	—	—	Good.
Ri.	1·5	10·8	11·6	2	915	—	—	Poor.
Ru.	2·5	10·9	11·8	2	747	102	25	Good.
St.	2	9·1	11·2	3	1309	—	—	Poor.
Shelt.	3	11·5	—	—	1222	—	—	Good.
Shep.	2	10·7	12·0	2	700	91	31	Good.
S.A. 1	2	9·4	13·3	8	700	—	—	Poor.
S.A. 2	4	9·7	11·6	12	—	92	31	Poor.
She.	5	12·0	11·5	7	1500	111	30	Poor.
Per.	4	11·8	—	—	1116	114	25	Good.
W.E.	6	10·9	10·2	14	2583	100	28	Fair.
B.	3	11·3	—	—	1093	90	31	—
Stant.	1·5	13·1	14·8	3	670	103	32	Good.
W.J.	3·5	11·4	13·6	7	1507	90	36	Poor.
N.	7	11·9	12·3	4	1680	104	28	Good.

at the thirty-fourth week. In the second pregnancy, when the patient had been given appropriate treatment, labour was again premature, but on this occasion at the thirty-seventh week. This fact is recorded, but obviously no conclusions can be drawn from it.

Hæmatology.

From the hæmatological point of view this investigation was prompted by the belief that folic acid was used quantitatively in human nutrition. If this was so, minor deficiencies should be evident amongst persons under nutritional stress, and this stress would be more commonly found in pregnancy. That this was likely was also suggested by a paper of Elsom and Sample (1937), who showed that macrocytosis could be readily produced in pregnant women by comparatively trivial dietetic restriction. They explained their changes on the basis of vitamin *B* deficiency, but we now know that deficiency of the then known vitamin *B* factors could not produce anæmia. It was thought that the factor most likely to be deficient was folic acid.

Wilson, Saslaw and Doan (1946) had shown experimentally in monkeys that lymphopenia was the most consistent sign of folic acid deficiency, so that it was felt that the examination of lymphocytes would be of value in deciding which patients were likely to respond to folic acid.

The purpose of the investigation was therefore to show that mild macrocytic anæmia was commonly present in pregnancy, that it would respond satisfactorily to the administration of folic acid, and that, finally, its diagnosis would be assisted by consideration of the absolute lymphocyte count.

Anæmia and its Response to Folic Acid.

The material consists of 16 patients who were selected on the previously mentioned grounds of either macrocytosis or failure to respond to iron therapy. One of them was observed during two successive pregnancies (which makes 17 observations in all), while three failed to report for a second blood check; this leaves 14 patients available for the judging of response to treatment. Volume determinations were performed on 10 occasions and yielded an average mean corpuscular volume of 100 cubic μ and an average mean corpuscular hæmoglobin concentration of 30%, confirming the presence of a mild dimorphic anæmia. Sternal puncture was performed on four occasions, but was discontinued as, in contrast to the situation in the more severe cases described by Sheila Callender (1944), it did not assist in the diagnosis. All patients were given folic acid (five milligrammes three times a day). The findings are summarized in Table I. It will be seen that the anæmia is mild, the hæmoglobin value averaging 10·9 grammes *per centum*; but despite this, an average gain of 1·6 grammes *per centum* of hæmoglobin was obtained, and this can be shown to be statistically significant. The average rate of gain was approximately 0·5 of a gramme *per centum* of hæmoglobin weekly and this was associated with great improvement in the patient's symptomatic condition. It can therefore be concluded that a mild macrocytic anæmia is commonly present in pregnancy and that it responds satisfactorily to folic acid.

TABLE II.

Hæmoglobin and Lymphocyte Values from 32 Consecutive Patients Routinely Checked at the Women's Hospital within the Last Three Months of Pregnancy.

Hæmoglobin Value. (Grammes *per Centum*.)	Number of Lymphocytes per Cubic Millimetre ×100.	Hæmoglobin Value. (Grammes *per Centum*.)	Number of Lymphocytes per Cubic Millimetre ×100.
11·3	34·5	12·0	37·4
10·4	25·2	13·5	24·7
13·5	19·2	12·5	27·2
12·0	19·3	14·1	51·4
13·8	21·8	10·3	56·2
12·7	31·6	10·4	30·7
10·4	32·0	11·6	14·2
8·7	24·9	11·6	33·9
15·1	23·5	10·6	32·2
15·5	43·7	11·6	15·8
11·6	26·4	12·5	32·2
12·5	30·1	13·0	22·8
12·7	15·7	12·5	32·0
11·6	73·3	11·6	69·4
12·5	39·6	13·5	16·4
13·0	18·6	6·4	7·6

Lymphocyte Levels.

Examination of the absolute level of lymphocytes in these patients showed it to be consistently low. This had been expected, but it was possible that lymphopenia occurred normally in pregnancy and therefore 32 patients from the Women's Hospital (Table II) were examined with regard to their lymphocyte level. These patients were attending for routine testing at approximately the same stage of pregnancy at which our patients were seen, and lymphopenia was found to be the exception. To exclude anæmia *per se* in pregnancy as a cause of lymphopenia, 15 consecutive patients (Table III) referred to a hæmatologist for anæmia of pregnancy of unspecified type were examined and failed to show a depression in the number of lymphocytes to the extent seen in our macrocytic series. The results are summarized in Table IV and the differences can be shown to be statistically significant.

Finally, if lymphopenia is really an index of folic acid deficiency, it should be directly related to the gain of hæmoglobin resulting from treatment. This can be shown

to be so, as a correlation coefficient of −0·68 was obtained between lymphocyte level and gain of hæmoglobin per week in the 12 cases in which evidence on these points was available.

It can therefore be concluded that the absolute lymphocyte count is of assistance in the diagnosis of these anæmias.

TABLE III.

Hæmoglobin and Lymphocyte Values from 15 Consecutive Patients Referred for Anæmia of Pregnancy of Unspecified Type.

Hæmoglobin Value. (Grammes *per Centum.*)	Number of Lymphocytes per Cubic Millimetre ×100.	Hæmoglobin Value. (Grammes *per Centum.*)	Number of Lymphocytes per Cubic Millimetre ×100.
13·3	12·3	13·1	17·7
11·3	10·9	11·2	7·7
13·0	20·7	10·5	9·8
11·2	27·0	12·3	22·0
10·2	23·4	11·8	6·6
11·7	11·7	10·8	13·2
10·9	20·2	12·3	18·1
13·4	13·3	—	—

Conclusions.

Taken at its face value, we believe that the improvement in symptoms and hæmoglobin levels must cast grave doubt on the conception of a physiological anæmia of pregnancy. We further believe that this conception has delayed the recognition of these cases, with consequent unnecessary morbidity for the mother and danger to the child. Again, the possibility of occasional dramatic relapse into the serious megaloblastic anæmia of pregnancy, so difficult to diagnose, makes early recognition of these cases of primary importance.

TABLE IV.

Comparison of Lymphocyte Levels in Patients with Macrocytosis, Patients Referred for Anæmia in Pregnancy and Patients Examined Routinely in the Last Three Months of Pregnancy.

Series.	Number of Cases.	Mean Hæmoglobin Value. (Grammes *per Centum.*)	Mean Number of Lymphocytes per Cubic Millimetre.	Standard Error of Mean Number of Lymphocytes.
Macrocytic ..	16	10·9	1135	130
Anæmic ..	15	11·8	1567	158
Routine ..	31	12·3	3148	260

In the diagnosis we feel that lymphopenia is of proven value, but it may not be present in the early stages and is likely to be confused by the inaccuracy of routine white cell and differential counts. Quite equal in value is the examination of the slide for anisocytosis by a competent hæmatologist, for, if we are correct in our views, each case must be assessed individually when symptoms of anæmia appear. By this we mean that anæmia can occur in pregnancy at hæmoglobin levels which are considered normal. With no hæmoglobin standards for comparison and with volume determinations invalidated by the inherent variability of routine red cell counts, there therefore appears no other alternative.

As nutritional deficiencies are most likely to appear in the last three months of pregnancy, we feel that the routine examination of patients for anisocytosis and lymphopenia at this stage would lead to an early recognition of those affected and a greater understanding of anæmias of pregnancy.

Acknowledgements.

We should like to thank Dr. Lucy Bryce for the hæmatology and for access to 15 counts from cases in which patients were referred to her for anæmia in pregnancy. Dr. R. A. Barter kindly made available material from 32 cases of pregnancy under routine check at the Women's Hospital, Melbourne. Dr. Kate McKay can be considered an instigator of this investigation, in that it was due to her courtesy that the first patient of our series was treated with folic acid.

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THE DOCTOR IN COURT.

By JOHN BOSTOCK and ATHOL H. ROBERTSON,
Brisbane.

THE importance of some knowledge of legal and court procedure cannot be under-estimated. At any time, in any year, a medical practitioner may have to give evidence in a law court, when his reputation may be at stake and bared to the view of the multitude and to the Press. If for no other reasons he should study legal procedure before the occasion occurs. Such prophylaxis is important.

It is the rarity of a court encounter which creates a great problem to the doctor. The legal world presents a point of view which is extraordinary to the medical mind.

It must never be forgotten that legal procedure, as practised in the court, is the result of evolutionary growth over thousands of years. Myriads of extraordinary situations have arisen; these have been discussed and judgements have been given and recorded. Thus there is an enormous background of recorded historical argument. It must be remembered that every conceivable form of rhetorical trickery which money could buy and wit provide has been employed to gain favourable judgements, so of necessity the law must provide criteria for the evaluation of evidence and a procedure which will offer a fair presentation of cases by all parties.

Too often the medical man criticizes the law instead of following the legal code.

It is our purpose to provide a guide for those who may occasionally be a player and not a mere spectator of legal encounters which are played in an arena as clearly defined as in a game of football, and with provisions as for offside, handling the ball, time, size of ball *et cetera*.

The medical witness has little opportunity of practising in the legal sport. A doctor in assessing the truth of a case uses every form of evidence, first-hand and hearsay. He sees the relatives and friends. He regards every whisper as having a significance which must be considered, whereas the law will not allow that hearsay evidence is valid, with the notable exception of the dying declaration.

What is Evidence?

The following is extracted from a well-known legal authority (Phipson, 1942) and is confined to what are considered relevant definitions and explanations which will assist the doctor in facing any cross-examination. Phipson implies that the law is not a science, and throughout the book it is stated that there are shades of meaning to some terms, whilst no satisfactory definition can be given to others. The following extract epitomizes his views:

The subject of evidence is not one which lends itself readily either to definitions or divisions. Few of its terms have acquired settled or unambiguous meanings, and no two writers adopt the same classification.

The following divisions are commonly employed in practice and comprise most of the relevant distinctions. (i) Evidence: This is the means by which the court is informed as to issues of fact and the subject matter of such means. (ii) Direct evidence: The thing (or facts) is produced in court or the testimony or declaration of someone who has perceived it. (iii) Indirect, circumstantial or presumptive evidence: Other facts are proved from which the existence of the given fact may be inferred. (iv) Real evidence: This is material evidence other than documents. (v) Primary evidence: This is "the best or highest kind of evidence"—for example, the production of an original document or proof of admission of its contents by the party against whom it is tendered. (vi) Secondary evidence: This is the testimony of a witness who has read the document. (vii) Hearsay evidence: Oral or written statements made by persons who are not parties and are not called as witnesses are inadmissible to prove the truth of the matter stated. A notable exception is a "dying declaration" in homicide, and then only in the case of the declarant. This declaration must be complete—unfinished declarations are inadmissible.

Let us now consider an illustrative case. Medical evidence, based on the sworn statement of a son and of a close friend, led to the certain diagnosis of senile dementia. No woman with such signs of mental, emotional and physical deterioration could possibly have produced a will dependent upon intimate knowledge of the nature of the act, the amount of her property, the implications of the will and the justice of her benefaction. In spite of reasonable medical opinion, the judge rightly found against the doctor, because his evidence was "hearsay". He had never seen the patient, whereas the solicitor who drew up the will had "direct evidence" that she was of testamentary capacity.

Further difficulties in the admissibility of evidence are neatly shown in the judgements concerning the proof of parentage. In the Preston-Jones case (*The Lancet*, 1950) the divorce commissioner considered that 360 days could elapse between coitus and the birth of the child. This judgement was subsequently upset by a decision of the House of Lords, Lord Simonds observing that the court had judicial knowledge of the normal period of human gestation.

It is apparent that there must be a legal standard until such time as medical knowledge becomes final, precise and irrefutable. Owing to the ever-widening perimeter of medical science, it is unlikely that this goal will be achieved in our lifetime.

Compulsion to Enter the Witness Box and Privilege.

Faced with the foregoing difficulties, the doctor may decide to evade giving any evidence at all. On this point the law is clear. The doctor can be compelled, as a competent witness, o attend the court and be sworn. The sole exceptions are sovereigns and foreign ambassadors. In the witness box, whilst he may claim privilege from some questions, as the answers may incriminate him, personally he is compelled to be sworn and give evidence.

Some matters protected from disclosure on the grounds of privilege are: (i) professional confidences; this privilege is reserved only for lawyers; (ii) title deeds, evidence of lien; (iii) matrimonial communications between husband and wife; (iv) criminating questions—forcing evidence against oneself. As will be shown later, a claim may be made at any stage and is determined by the judge. When allowed, it protects the witness from further answers to the relevant question.

The attitude and morale of the doctor during his periods as a witness will partly depend on whether he is called as a defendant in an action against him or as an incidental and perhaps important expert witness.

Your Evidence Must be Correct.

Just as the sportsman must prepare for an important event, the doctor should consolidate his position before going to court. The following points are important.

1. Read and reread your notes. If they are at all illegible, have them typed.

2. Make certain as to dates, conversations over the telephone, or other items which are not specified in the notes.

3. A conference with counsel in his chambers, and usually with the instructing solicitor present is advisable. Here the pros and cons of the case are discussed and the importance of the doctor's evidence is determined. A transcript of previous evidence may be summarized and the opposing counsel's strategy and tactics emphasized. Then a statement of relevant facts is taken from the doctor to consolidate his observations and for counsel's use in cross-examination.

The Witness Box.

Lawyers are usually most considerate of the disruption caused to the doctor's practice by his enforced absence and endeavour to call him so that waiting in the precincts of the court is as short as possible. This waiting period should be utilized by thinking along the following lines. (i) I know more about medicine than the opposing counsel. (ii) I will stick to facts. (iii) I will refuse to be ruffled. (iv) I will take my time in answering questions.

The objection of "confrontation" or the production of witnesses in the box is not only for cross-examination, but also to enable the court and parties to observe their demeanour, so that a calm, accurate and succinct witness is an asset.

Having been sworn, the doctor faces the judge and must realize his key position in the proceedings.

Court Procedure.

The duties of the judge are important and diverse. He must (i) decide all questions as to admissibility of evidence, (ii) instruct the jury, (iii) determine at the close of the trial whether any evidence has been given fit to be considered by the jury, (iv) explain to the jury the issues and sum up; in the summing up he may express his own opinion to the jury.

Let us now consider the matter more closely in relation to the admissibility of evidence.

In all cases tried before a jury the counsel must endeavour to influence it directly. As the members are human, he endeavours to influence them by suggestion and persuasion, by pathos or humour, by the tragic or the bizarre. This results in frequent efforts to elicit evidence which may not be strictly relevant.

As in a game of football, the competitors try to evade the rules and get past the referee, who in this case is the judge; the participant—the doctor—however, is at all times permitted to ask His Honour whether he must answer the question.

Thus one of us was once asked (i) about the inner working of his medical practice, (ii) about a conversation with another patient about his own affair, (iii) whether a certain person had been his patient. On His Honour's being asked if it was necessary to give evidence, he disallowed the first and second questions as inadmissible. On the third occasion he was sent out of the court whilst the counsel discussed whether the question was essential to the progress of the case.

The Cross-Examination.

The doctor now faces the planned questioning by counsel, the logic and pattern of which are not usually apparent, but which frequently commences with a discussion of his professional training and experience.

At the conclusion of the examination by the opposition, the doctor may feel that he has been allowed only partly to express his facts, but he must remember that his

counsel has yet to speak, and when he does the very points the doctor desired to elaborate will be asked and often hammered home.

Much embarrassment will be saved if certain standard replies are used. Verbosity is dangerous and opinions should never be volunteered. "Yes", "no", and "I cannot answer that", given after due consideration of the question put, will answer the majority of questions.

Many tactics of counsel, such as *sotto voce* remarks, apparently idle comments during the ruffling of paper or books, are used in the examination, and the witness may give an unconsidered reply, often believing the question not part of the procedure. This is not so—these are deliberate tactics.

One of us succumbed to this trick when counsel mentioned a lapse on his client's part, as if it was a matter of no importance. Witness agreed that the lapse occurred, and the evidence read that it was admitted that the client had one lapse. His other numerous relevant lapses were not discussed and so were not recorded as evidence.

As in sleight of hand, an incident passes so quickly that it is only later that one appreciates what has happened.

In the process of law there is neither virtue nor vice, neither loyalty nor disloyalty; it is a struggle for victory in facts.

If directed by the judge to answer a question to which witness has replied "I cannot answer that", witness may say: "Can you put the question another way?" If counsel refuses, he must answer as best he can.

If the doctor is a principal in the case he should note any medical untruth or flaw in counsel's statements and answer so as to indicate the latter's lack of knowledge of that one detail.

On Authorities.

During cross-examination counsel may produce impressive medical text-books and, picking one up, read a passage and ask the doctor to discuss the passage, perhaps asking whether he agrees or not with such an authoritative statement. Beware! The book may be completely outdated or written by a medical "crank", and before answering, the witness should ask counsel to mark the passage and give up the book so that the date, author and context may be read by him. Similarly, check statements read from clinical notes, and often the impressive barrage of quoted authorities will cease abruptly.

The doctor may be asked whether he agrees with an "opinion" of a colleague or "authority" on some point, clinical, physiological or pathological. Often this answer is one which returns like a boomerang at a critical point in the case to wound him who gave it. Think hard! The answer may be clearly "yes" or "no"; but if not, witness may well say: "I cannot answer that until I have examined the basis for P's opinion."

A display of erudition, correct or conjectured, is usually dangerous and best avoided unless the doctor is called in the role of the "expert witness". The latter is one called to give evidence on technical aspects of a case and indicate his opinions thereon. It is important to realize that the expert opinion is not always highly regarded in law courts, though in certain specialties men such as Sir Bernard Spilsbury established the highest reputation for accuracy, logic and relevance of evidence and opinion.

Expert's evidence is summarized by Phipson as "of slight value—they are proverbially biased in favour of the side which calls them, as well as over-ready to regard harmless facts as confirmation of preconceived theories".

Cross-examination may elicit that the expert was not in a fit state of mind or health to form an opinion, or is interested or corrupt, or has expressed a different opinion at other times. Thus the medical expert must have a character comparable with Cæsar's wife. This is difficult.

Conclusions.

Whilst the foregoing summarizes some of the present-day procedures concerning the giving of medical evidence in courts, it is probable that in the future there may be changes. Through the years there has grown a technique whereby truth may emerge with more clarity from the welter of differing and conflicting evidence. Modern research in psychoanalysis has shown that it is sometimes possible by new methods to obtain direct evidence from an individual, without the conscious barriers leading to prevarication and evasion.

There are machines which indicate reactions whereby one can tell whether the person is telling the truth. Glenn D. Higginson (1950) quotes two American cases in which the "lie detector" has been used in the courts, and its success was of considerable assistance in determining the credibility of the witnesses. There are authorities who believe that this victory presages a great future for the "lie detector", and they hold that it will be as acceptable in court as finger-print testimony.

Whilst such a tremendous innovation must have great opposition, there are many who consider it will have its uses, particularly when the distinction between truth and falsehood is so fine that other methods fail to detect the line of demarcation. Until this millennium arrives, the doctor in court must follow the age-old procedures of traditional law. If he is wise he will regard the law as remarkably efficient social machinery for the preservation of our rights and freedoms.

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Reports of Cases.

A CASE OF TOADFISH POISONING.

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THERE are few cases on record in Australian literature of death from eating the poisonous toadfish—no doubt owing to its repulsive appearance. The genus is well represented in our seas—particularly in the south—and the following case illustrates the highly potent nature of the poison which it carries.

Clinical Record.

On December 6, 1950, a boy, S.C.M., aged eleven years, living at Castle Forbes Bay, was given five small fresh fish which had been headed and cleaned but not skinned. He took these fish home and his mother cooked four of them, the cat having stolen one. The fish were placed before S.C.M. and his father, but the latter, after tasting a small portion, decided he did not fancy the fish and he gave his share to the cat. S.C.M. asked his mother to cook his two fish a little longer, after which they were eaten at 6 p.m.

Tea was concluded at 6.20 p.m. and the boy went on his bicycle to do a message. When he returned at 6.40 p.m. he complained of feeling "queer"; his legs, feet and hands were numb and his body seemed to be "floating". Within five minutes he complained of feeling cold and said he could not move his arms or legs. His lips felt stiff. He was given an aspirin, but could not swallow it. The boy had lost consciousness by 7.30 p.m. and was moribund when examined by Dr. H. P. Coats at Huonville at 7.50 p.m. He died soon after 8 p.m.

Post-mortem Findings.

Apart from some pulmonary œdema and some congestion of the organs, no abnormality was detected. The routine examination of histological sections did not assist in any way.

Further Investigation.

The fish immediately came under suspicion, in spite of the father's statement that they were "mountain trout". The suspicions were strengthened by the action of the family cat, for at 9.30 p.m. it was seen to be paralysed in the back legs, and it was found dead in the morning.

A warning was issued to parents in that area, and Hobart detectives, using explosives, were able to obtain some more of these fish from the same tidal creek. They were identified by Dr. Pearson, of the Tasmanian Museum, as belonging to the family Tetraodontidæ—puffer fishes having the teeth of each jaw coalesced into two tooth plates. The genus is abundant in species and numbers. The species in this case, *Spheroides liosomus*, is fairly common in Tasmanian waters and sometimes enters tidal creeks, possibly for spawning.

Aqueous extracts were made from several organs of the fish as well as from the stomach contents of the boy and the cat. All were found to contain a potent and fast-acting toxin when injected subcutaneously into guinea-pigs. A few drops of an aqueous extract of toadfish skin caused paralysis and death in a small guinea-pig in a matter of minutes.

Police inquiry revealed a further interesting fact. The day before the tragedy three similar fish were caught and fed to three cats belonging to another family. The cats soon became sick, vomited, and later died. Some hens picked at the vomited material, and as a result one died and two were seen to be paralysed. (It is not recorded whether anyone ate the dead fowl.)

Discussion.

According to Gudger (1930), ichthyotoxism—fish poisoning—may occur in three ways: first, by the infliction of wounds by means of spines which have attached thereto glands secreting poisons; secondly, by means of a poison found in the blood plasma of the fish; and thirdly, by ingestion of poisons found in the tissues of the fish, either ptomaines produced by decomposition or toxins secreted by the tissues. The first two poisons enter by injection, the last by ingestion.

Phisalix (1922) lists 65 fishes as having poisonous stings, 14 having poisonous blood, and 121 species that are poisonous to eat.

Toxins in the tissues of a fish would appear to be formed either by the fish itself or from substances eaten by the fish. A wide variety of such substances are listed—for example, marine snails and plants, corals, jellyfishes, worms, metal contaminants, and even the manchineel tree, the fruit from which drops into the sea. Somner and Meyer (1941) have shown that mussels and clams caught off the Californian coast sometimes become poisonous owing to ingestion of a certain type of plankton. The poison is an alkaloid belonging to the class such as strychnine, muscarine and aconitine, a few milligrammes being a fatal dose for man.

While some of these poisonous fish are repulsive and unlikely to be eaten, others appear quite edible. Lee and Pang (1945) reported outbreaks of fish poisoning in Honolulu from eating sea bass and red snapper.

The variability of the toxin in some species is indicated by the following tentative conclusions reached by Mann (1938). (i) Toxic fish are present in certain well-defined areas. (ii) The same species may be non-toxic in other areas. (iii) There is a seasonal influence on the toxicity. (iv) Individual fish of the same species caught in the same area on the same day vary in their toxicity. (v) The symptoms are caused by a specific toxin, the nature of which is undetermined. (vi) The symptoms are so severe that all visitors should avoid eating any fish caught in the Culebra-Virgin Island area.

Professor J. B. Cleland (1942) reports illness from eating the "Stink Fish" and the "Chinaman Fish".

The following extract from an article also by Cleland (1912) shows that fish poisoning was well recognized at that time.

> *Fish Poisonous to Eat.* Very few references to poisoning by fish, apart, that is, from putrid or bacteriologically contaminated fish, fresh or tinned, occur in Australian literature. The following appears in Afialo's "A Sketch of the Natural History of Australia", 1896, p. 247. "The poisonous toads (*Tetradon*) and porcupines (*Diodon*), relatives of the huge but harmless sunfish (*Orthagoriscus*), have often caused accidents to those careless or ignorant enough to eat them. Some lads were poisoned in this way at Coogee not many years ago, and a family of three died early in the century from the same cause."
>
> I am indebted to Mr. A. MacCulloch of the Australian Museum for the following reference (Richardson, Zool. Erebus and Terror, Fishes, p. 63, who quotes Mrs. Meredith, Notes of New South Wales, London, Murray, 1844, p. 155). Writing on the toad fishes, *Spheroides hamiltoni*, Richardson, from Port Jackson, he says: "They are highly poisonous . . . I know one instance at least of their fatal effects; a lady, with whose family I am intimate, having died in consequence of eating them."
>
> According to the *Australian Medical Journal*, two boys died at Randwick in 1871 from eating some cat fish caught in Coogee Bay and cooked on the beach. They walked home in great suffering and died almost immediately.

In Ross's "Hobart Town Almanac" for 1832 there appears an illustration of "The Poisonous Toad Fish", together with an article by colonial surgeon James Scott, in the course of which he makes the following statement:

> The melancholy and dreadful effect produced by eating it was lately instanced in the neighbourhood of Hobart Town on the lady of one of the most respectable merchants and two children, who died in the course of three hours, without being able to give any notice of their danger. The poison is of a powerful sedative nature, producing stupor, loss of speech, deglutition, vision, and the power of the voluntary muscles, and ultimately in entire deprivation of nervous power and death. At the inquest over the above bodies, the effect of poison was satisfactorily proved by giving part of the fish left by the unfortunate individuals to two cats, which soon became affected. When both were in a dying state, one had 25 drops of the arsenical solution introduced with a silver tube into the stomach, and rapidly recovered, while the other, which was allowed to take its chance, quickly died.

Further particulars of the case are given in *The Colonial Times* of March 29 and April 5, 1831.

The Japanese have made an intensive study of toadfish poisoning. The Tetrodons, called by them *Fugu* fish, are abundant in species and numbers in the waters of Japan and are, generally speaking, very poisonous. Savage (1920), reported by Robinson (1937), states that over 700 people in Japan died from eating this fish in the seven-year period between 1885 and 1892. The toxin—*Fugu* toxin or tetrodontoxin—is very abundant in the breeding season. Tried on dogs, it was found to paralyse those nerve centres which control sensibility, motion, circulation, and respiration. The Japanese conclude that the toxin is a toxalbumin rather than an alkaloid. It is not destroyed by prolonged heating.

Summary.

A case of toadfish poisoning is described and references are made to some other cases of fish poisoning and to the mechanisms by which the poisoning is produced.

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Reviews.

PATTERNS OF DISEASE.

"PATTERNS OF DISEASE", by Frank L. Apperly, is based on the author's lectures on pathology in the Medical College of Virginia.[1] It is not, however, nor is it intended to be, a text-book of pathology in the ordinary sense. In his preface, Apperly voices the opinion that while standard text-books of pathology are essential for the morbid anatomist and the histopathologist, they are unsuitable for the "overburdened medical student" and the young doctor in practice, because they include too much description of structural detail and too little discussion of function and of disease processes. His book barely mentions histopathology and contains no illustrations of microscopic structure. It is an attempt at simplification, but on the whole not a successful one. In his preface the author admits that in many places he has "exhibited a shocking dogmatism . . . in order to avoid lengthy and tortuous arguments which can be of interest only to the investigator and the more advanced physician". What is the result of all these omissions and attempts to make difficult things easy? A book of 446 pages, which is not particularly easy to read since it lacks the precision, orderly arrangement and completeness which are essential to a scientific text-book. It is disjointed; the style is often awkward, verbose and careless, suggesting hasty writing and little revision. The lectures on which the book is based are probably intended to be informal and colloquial. Such lectures, illustrated by the line drawings and charts of the book, may prove a useful adjunct to, but are not a substitute for, systematic instruction in pathology.

FIFTY YEARS OF SCIENCE.

ALMOST every branch of science has recently in its appropriate journal made an assessment of the progress achieved during the first half of this century. In "Scientific Thought in the Twentieth Century",[2] under the editorship of Professor A. E. Heath, an attempt is made to present the reader with a compact survey of the whole field. Professor Heath has chosen his team well, and the limits of space he has imposed will be approved. The separate chapters are devoted to the philosophy of science, statistics, astronomy, physics, chemistry, geology, zoology, genetics, general medicine, social medicine, neurology, psychology, social anthropology and sociology. Just why biochemistry and botany should be left out is puzzling. The first chapter is devoted to the philosophy of science and has been placed in the hands of a philosopher. Men of science have an uneasy feeling that when one of their number becomes interested in the philosophy or history of his subject he has ceased to be an experimenter. However, those who study this subject will find an able treatment. Possibly first place will be given by the reader to the article on astronomy by the Astronomer Royal, who has remained faithful to the high traditions of exposition established by his predecessors. General medicine, social medicine and neurology are all admirably handled by experts in each subject. The chapter on physics may well bewilder the reader by the rapidity of change in theory in recent years, for the atom is now a complex of protons, neutrons, mesons, negative electrons, positive electrons and electrons which rotate. In a few years' time the picture will assuredly be altered towards either simplification or the reverse. In chemistry the more recent conception of valency depending on shared electrons is well set out. An earnest attempt is made to present statistics without over-much mathematical elaboration, but even so there is some hard reading. In social anthropology the vigour with which older views are assailed will make the reader wonder whether present doctrines will not be similarly attacked in a few years. It is a pity that in the chapter on psychology several statements are made which suggest unmerited criticism of medical theory and practice; the writer, Sir Cyril Burt, is aggrieved that medical psychiatrists have been entrusted with the diagnosis of defective children and makes some cutting remarks about the school doctor. The retort is obvious that a doctor ignorant of psychology will make fewer mistakes than a psychologist ignorant of medicine. Except for this and the exclusion of biochemistry and botany the book can be warmly commended.

ELECTROENCEPHALOGRAPHY IN CLINICAL PRACTICE.

"ELECTROENCEPHALOGRAPHY IN CLINICAL PRACTICE", by Robert S. Schwab, of the Massachusetts General Hospital, is stated by the author to be intended for neurologists, internists, psychiatrists and neurosurgeons, and in no sense to be a manual for electroencephalographers.[1] This aim has been maintained in a series of clearly written chapters aided by generous illustrations with clear legends. A practical attitude is sustained throughout in sections dealing with history, neurophysiology, technique, the normal and the abnormal electroencephalogram, epilepsy and other conditions, and psychiatric disorders. The value of electroencephalography in research is touched upon, and information given on the staffing of the laboratory, its cost and what may be charged the patient. At the end comes a glossary and a useful index.

The author attempts to put forward the values of electroencephalography without over-emphasis. Thus he remarks that to the neurologist it is usually but a method of confirming organic disease and its localization which may, except in a small percentage of cases, be more exactly confirmed by air studies or by arteriography. Its chief use to the psychiatrist, however, is to indicate the presence of organic disease and the need for a neurological colleague. But even so a negative result, particularly in vascular disease, may not help greatly. To the neurosurgeon it is an accessory means of localization of cerebral tumour, which is 73% correct against 90% for air studies. As 90% of brain tumours show abnormal electroencephalograms, as against 20% of vascular lesions after four days if the author is correct, its value here should be great.

However, the study of the relation of electroencephalography to vascular lesions is perhaps the least satisfactory part of the book, and requires amplification. Indeed an explanation might be given as to why the area affected in primary vascular lesions causes no abnormal electrical discharge, whereas about an electrically inert tumour a similar vascular disturbance is suggested as the source of the slow waves. Nevertheless, the author is to be congratulated on a clear and useful book, which will be of value to the clinician interested in neurology.

POISONS: THEIR ISOLATION AND IDENTIFICATION.

SINCE Bamford's book "Poisons: Their Isolation and Identification" was first published in 1940, two further editions have been produced, and this present book, the third edition,[2] will serve further to stimulate interest in toxicological analysis.

The published work dealing with poisons, their characteristics and identification is meagre indeed, but this book is a substantial part of it. The book is essentially practical and describes the chemical methods available for the identification of all the usual poisons, including also the more recent drugs such as the antihistamines and the synthetic analgesics. It is, perhaps, a pity that no mention is made of biological tests, since these are sometimes more specific and more sensitive than some of the chemical tests

[1] "Patterns of Disease on a Basis of Physiologic Pathology", by Frank L. Apperly, M.A., M.D. (Oxford), D.Sc. (Melbourne), F.R.C.P. (London); 1951. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 470, with 50 figures and 37 charts. Price: 8s. 6d.

[2] "Scientific Thought in the Twentieth Century: An Authoritative Account of Fifty Years' Progress in Science", edited by Professor A. E. Heath, M.A.; 1951. London: Watts and Company. 8½" × 5½", pp. 404. Price: 42s.

[1] "Electroencephalography in Clinical Practice", by Robert S. Schwab, M.D.; 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 6½", pp. 206, with 106 figures. Price: 61s. 9d.

[2] "Poisons: Their Isolation and Identification", by Frank Bamford, B.Sc.; Third Edition, revised by C. P. Stewart, D.Sc., Ph.D., with a foreword by Sir Sydney Smith, C.B.E., M.D., F.R.C.P.; 1951. London: J. and A. Churchill. 8½" × 5½", pp. 324, with 23 illustrations. Price: 25s.

commonly employed; the biological methods for the identification of curare, atropine or picrotoxin, to name only a few drugs, could hardly be equalled for sensitivity.

"Bamford" is a conveniently small laboratory handbook, and as such is a most valuable book to those engaged in the study of toxicology and the chemistry of drugs. Some of the more useful references are given as page footnotes, and although a more extensive bibliography would be an advantage, the book provides sufficient information to enable a literature search to be often relatively unimportant. Dr. C. P. Stewart is to be congratulated upon a satisfactory revision of a very useful book.

CHLOROFORM ANÆSTHESIA.

"WE certainly do not advocate any widespread revival of the use of chloroform. On the other hand, we can scarcely concur in the marked fear of the agent which seems to prevail at present." Thus Ralph M. Waters and his associates on pages 71-72 of their small book "Chloroform: A Study after 100 Years".[1] With this general conclusion all competent anæsthetists and well-informed surgeons must agree.

The work is the result of about ten years of laboratory, clinical and statistical investigation, directed to the reassessment of chloroform as an anæsthetic agent in the light of modern knowledge and practice. It represents an exhaustive comparison of 1111 expert administrations of chloroform with the use of other agents in 36,968 cases. The data submitted indicate that, under such good conditions, liver damage from chloroform is not much greater than that caused by other agents, that its effect on renal function is negligible, and that cardiac irregularities may be even less frequent than with cyclopropane or trichlorethylene.

A serious depression of myocardial function, however, is shown to be the special prerogative of chloroform. It affords a far too narrow margin, even with the best control of dosage and administration, between safety and danger. Although there was no immediate mortality in this series, grave cardiac disturbances occurred in several cases, in some of which warning was given only by continuous electrocardiographic recordings. Even if prompt restorative measures, notably the inflation of the lungs with oxygen, were successful in these alarming circumstances, recourse to chloroform would seem to be incompatible with ordinary prudence.

This book is very well written, although rigid condensation and the American idiom at times cause some obscurity. The final chapter by Waters himself is a model of clarity and brevity. He insists that chloroform does not deserve to be abandoned as a surgical anæsthetic, but admits the need for a high order of skill and the very best of facilities in its use. The work concludes with an imposing list of references and a satisfactory index. It should be read by all interested in good anæsthesia, especially those who would revive the common and indiscriminate use of chloroform.

OPHTHALMIC DIAGNOSIS AND TREATMENT.

THE "Encyclopædia of the Eye", by Conrad Berens and Edward Siegel, is a small book written for those who desire a ready reference to the diagnosis and treatment of the more common ophthalmic conditions.[2] It should be useful to general practitioners, medical students, ophthalmic nurses and social workers.

The subjects are presented alphabetically in sections that can be read in a few minutes—but each is authoritative, concise and clear. The longer sections have cross references to closely related subjects. References to publications are given only occasionally. Black and white or coloured plates diagrammatically illustrate some of the diseases most frequently seen. A detailed index covers thirty-five pages.

Australian ophthalmologists will be surprised that no differentiation is made between rubella and morbilli in describing the abnormalities caused during early pregnancy. These are simply attributed to maternal measles. Nevertheless, the book adds further credit to its renowned authors.

[1] "Chloroform: A Study after 100 Years", edited by Ralph M. Waters; 1951. Wisconsin: The University of Wisconsin Press. 9" × 5½", pp. 152, with 35 figures. Price: $2.75.

[2] "Encyclopædia of the Eye: Diagnosis and Treatment", by Conrad Berens, M.D., F.A.C.S., and Edward Siegel, M.A., M.D.; 1950. Lippincott: Philadelphia, London, Montreal. 8" × 5", pp. 272. Price: £2 13s. 9d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Liver Disease"; consulting editor, Sheila Sherlock, M.D., F.R.C.P.; editor for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch.; 1951. London: J. and A. Churchill, Limited. 8" × 5½", pp. 262, with 112 illustrations. Price: 25s.

An edited and illustrated record of an informal international conference on liver function and disease held at the Ciba Foundation, London, in 1950.

"Studies of Undernutrition, Wuppertal 1946-9"; Privy Council Medical Research Council Special Report Series, No. 275, by members of the Department of Experimental Medicine, Cambridge, and associated workers; 1951. London: His Majesty's Stationery Office. 10" × 6½", pp. 418, with 62 plates and 14 figures. Price: 12s. 6d.

A study in the effects of food shortage in Germany.

"Progress in Neurology and Psychiatry: An Annual Review", edited by E. A. Spiegel, M.D.; Volume VI; 1951. New York: Grune and Stratton. 9½" × 6½", pp. 576. Price: $10.00.

Designed not only for the specialist, but also for other interested practitioners.

"Transactions of the International and Fourth American Congress on Obstetrics and Gynecology", edited by George W. Kosmak, M.D., sponsored by the American Committee on Maternal Welfare, Incorporated; 1951. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10½" × 7½", pp. 840, with 152 plates and many text figures. Price: £7 1s. 9d.

Papers and discussion from members of congress representing many countries.

"The Medical Clinics of North America"; 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Mayo Clinic Number. 9" × 6", pp. 284, with 35 illustrations. Price: £7 5s. per clinic year (cloth binding) and £6 per clinic year (paper binding).

Contains a symposium on diseases of the kidney (20 papers) and articles on the relationship of arterial oxygen saturation to cyanosis, the familial incidence of Paget's disease of bone and the relation of sedimentation rate to age.

"Medical and Physical Diagnosis: Interpretation of Findings", by Samuel A. Loewenberg, M.D., F.A.C.P.; Eighth Edition; 1951. Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 10½" × 7", pp. 1264, with 717 illustrations, 41 in colour. Price: £7 5s. 3d.

Describes methods of clinical examination and discusses the findings of these and related special investigations in relation to diagnosis.

"The Management of Fractures, Dislocations and Sprains", by John Albert Key, B.S., M.D., and H. Earle Conwell, M.D., F.A.C.S.; Fifth Edition; 1951. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10½" × 7½", pp. 1232, with 1195 illustrations. Price: £8 8s.

A completely revised edition.

"Proceedings of the Third International Congress of the International Society of Hematology", Cambridge, England, August 21-25, 1950; edited by Carl V. Moore, M.D. (editor-in-chief); 1951. New York: Grune and Stratton. 10½" × 7", pp. 610, with 104 illustrations and many text figures. Price: $10.00 (cloth bound) and $8.00 (paper bound).

Complete papers and abstracts of papers presented by workers in all fields of hæmatology.

"Wiederherstellungschirurgie des Gesichts", by Professor Dr. Reinhard Perwitzschky; 1951. Berlin: Walter de Gruyter and Company. 9½" × 7", pp. 240, with 176 illustrations.

A book on the plastic surgery of the face written in German.

The Medical Journal of Australia

SATURDAY, NOVEMBER 17, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

SECURITY AND FREEDOM.

SOCIAL SECURITY was discussed in these columns in December, 1941, during the early days of the Second World War. In that discussion it was stated that to some people social security would mean an assured position in society for themselves, and equal provision of food and clothing with shelter not only in the heyday of strength and health, but in times of sickness and old age. The opinion was expressed that this subjective view was futile and soul-destroying. Social security was stated to be something which concerns every member of the community. It is not a passive state to be accepted as a right without any deserving. It is an active condition, to be earned, to be sought, for others as much as for self. Reference was made to an article by Oswald Stein in *The International Labour Review* of September, 1941. Stein was chief of the Social Insurance Section of the International Labour Office. He set out the object of social security in the following words: "To prevent those losses of productive life and capacity that can be avoided; to ensure that each makes the best possible use of his powers and aptitudes; to underwrite equality of opportunity with a real collective guarantee, taking effect in the event of an involuntary failure of individual effort, and in the attainment of those ends, to accord due respect to the supreme and inalienable values of individual dignity and liberty." Stein also declared that the essence of social security from one point of view was "the general and rational economy of manpower, yielding the greatest good for the greatest number". It is clear that some limitations to social security must be set. To wish to create a society free from individual risk and responsibility would be to accept a definition of social security which has already been described as futile and soul-destroying. If we accept the other definition of social security that has been given as an active condition to be earned, to be sought for others as much as for self, we recognize that each man carries a responsibility which he must accept. This was made clear in a recent discussion in these columns on professional freedom and professional responsibility. It will be remembered that T. F. Fox in his Croonian Lectures quoted Bernard Shaw's statement that liberty means responsibility and that that is why most men dread it. Accepting this point of view, we are forced to consider how far security and freedom can go hand in hand.

Light is thrown on this question by a small book recently published by John Macmurray, Professor of Moral Philosophy in the University of Edinburgh. The book, "Conditions of Freedom", comprises a series of three lectures delivered at the Queen's University, Kingston, Ontario, in 1949. Macmurray is concerned with what he calls the relativity of freedom. Before he explains what he means by this he points out that in the crowded history of modern achievement there is a wealth of good things that have been made available to mankind for ever. All these have their roots in freedom and this is our most precious achievement. It is not our power either of technique or of knowledge which matters; it is our faith in freedom. If that faith is lost, Macmurray believes that all is lost. The ancient and widespread belief that the supreme good of human life is happiness is, he believes, false. Freedom has a higher value than happiness. There is a sense in which freedom is absolute. Absolute freedom is simply our capacity to act, to form an intention and to seek to realize it. To act is to be free. This means that we are concerned about the future—the future is the field of freedom. Macmurray states that the idea of the absoluteness of freedom is not satisfying. He points out that as soon as we move towards a more concrete statement, the relativity of freedom appears and with it the paradox of freedom. The simplest expression of the paradox lies in the difficulty which we find in being ourselves. Our human nature eludes us. We are, and yet we are not, ourselves and in this is our freedom. "So freedom is at once absolute and relative: absolute, because if we were not free we should not be human at all; relative, because this freedom lies always beyond our present achievement as the goal of our existence." Macmurray believes that we flatter ourselves too much when we imagine that we love freedom and strive wholeheartedly towards it. There are few things which we fear so much. The idea of freedom is most attractive, but the reality is another matter. To act freely is to take a decision and to accept the consequences. The free man is the man who takes responsibility for his own life before God and his fellows. This brings us back to Fox's quotation from Bernard Shaw. Macmurray asks whether it is any wonder that when we are faced with the challenge of freedom our fear is usually more than a match for its attractiveness, and that we seek for the most part to escape the demand that it makes upon us. Macmurray thinks that our capacity to deceive ourselves in this matter is of extreme subtlety. If we are honest with ourselves most of us will agree that on many occasions we have been successful in self-deceit. The paradox of freedom thus is that we are free to choose between freedom and security. Macmurray holds that the choice is not voluntary, nor is it once for all. It is compulsory and it is perpetually recurrent. There is an element of illusion about it too. The demand for security is the reflection of

our fear, while freedom is the expression of our own reality. "If we use our freedom to escape from freedom we frustrate ourselves: if we persist in this choice we destroy ourselves." If we aim at security, Macmurray thinks that we aim at the impossible and succeed only in multiplying the occasions of fear and magnifying our need for security. There is no security for us except in choosing freedom, for our insecurity is our fear and to choose freedom is to triumph over fear. From these arguments it will be clear that an increase in freedom implies an enlargement of the field in which we can do as we please. If we wish to increase our freedom, we must remove obstacles which prevent us from doing as we like. In this regard Macmurray makes a statement which is obviously true, but which will not be very popular. He states that we can increase our freedom by limiting our desires without any change in the means of action at our disposal. "The free man is a man whose means are adequate to his ends."

One factor which cannot be excluded from any discussion on either security or freedom is the interdependence of human beings. We know that "no man liveth unto himself" and also that "we are all members one of another". This was made quite clear in the recent discussion on Fox's Croonian Lectures; it was also the main theme of our discussion on freedom and the spirit of man in these columns some three or four years ago. If a man is not willing to curb his own desires and thus to bring freedom more easily within his reach, he will be denied real freedom unless he realizes that his wishes for freedom cannot be considered apart from those of other people. At this point reference to service must be made. When we try to do things for other people we are helping them on the road to freedom and so to security. They receive freedom in these circumstances as a gift from us. We in like manner receive freedom from other people. Macmurray puts this very well. He writes: 'To believe in freedom, in any sense worthy of consideration, is to believe in setting other people free." If we do this we shall clearly, at times, have to sacrifice our own freedom. In regard to this reciprocity Macmurray writes: "In giving freedom to others, we have a right to hope that they in turn will have the grace and the gratitude to give us ours. But of this we can have no guarantee."

The conclusion of the matter is that the extent to which we achieve freedom in life depends upon the extent to which fear has been removed from it and also upon the extent to which the cooperation it demands "is positively or negatively motived". Security is something to be fought for and won, and it is to be won chiefly by efforts to secure the future of other people. If we are concerned with the security of others we shall have less time for our own fear. This does not mean that we are to have no concern about our own welfare. The point is that we (if we use the word to apply to members of the medical profession) should put our care for others in the forefront. The practice of medicine demands a life of service and, with the ideals of medicine always before him, the medical practitioner should not have great difficulty in striking a right perspective. When we think of security for mankind as a whole, we have to remember that the exercise of the faculties strengthens them and that atrophy follows disuse. If man is so secure that he has no need either to toil or to spin and is content with that state he will surely degenerate. The fantasy may be extreme, but H. G. Wells in his well-known story "The Time Machine" shows man in a state of physical and mental deterioration some 400,000 years hence. This exaggerated flight of the imagination is a parable of no mean order and should be read as such, for it is a fearful warning. Only the fringe of this vast subject has been touched upon. It is one which will repay careful study. Fox's Croonian Lectures were recommended previously to readers of this journal. A similar recommendation may be given in regard to Professor Macmurray's book "Conditions of Freedom".

Current Comment.

RECURRENT PTERYGIUM.

PTERYGIUM is a relatively common condition in this country, and Australian ophthalmic surgeons are familiar with its obstinacy. This applies especially to Queensland; so there should be considerable interest in the report in this issue of an investigation by J. B. Gilchrist Gibson of recurrent pterygium at the Brisbane General Hospital. The exact ætiology of primary pterygium remains elusive, although the role of a hot, dry, dusty climate is now generally accepted. Some three years ago, Arthur D'Ombrain,[1] of Sydney, in drawing attention to a "brilliant paper" by Sabri Kamel on the nature of pterygium, expressed agreement with Kamel's view that pterygium is an irritative disease due to exposure and not primarily a degeneration, but he stressed the fact that it is secondarily a degeneration. D'Ombrain is strongly of the opinion that pterygium and pinguecula are essentially one and the same process, ætiologically, anatomically and pathologically. He states that the stroma or subconjunctival portion of the pterygium presents a similar histopathological picture to that of pinguecula—namely, an extensive aggregation of fibrous tissue containing numerous elastic fibres and patches of amyloid and hyaline degeneration; but when this contracting fibrous tissue becomes anchored at one end to the unyielding corneal tissue, the looser conjunctival tissue becomes pulled towards the cornea. The cornea itself becomes involved in the inflammatory process and also in a secondary degeneration, with the presence of elastin, down to and including Bowman's membrane. When examined with the slit lamp, pterygium and pinguecula present the same appearances. The only essential difference between the two, in D'Ombrain's view, is one of site—depending on whether they are, or are not, in juxtaposition to the corneal limbus. Those that are not in contact, or do not come into contact, with the limbus remain pinguecula, but those that do so progress to formation of pterygium. This assumption is the basis of D'Ombrain's operation for removal of the pterygium, an operation which, according to H. B. Stallard in the second edition of his book "Eye Surgery", will probably replace all transposition operations practised in the past. D'Ombrain carefully shaves the head of the pterygium from the cornea (a thin layer of which he includes), dissects and excises the subconjunctival part of the pterygium from the under surface of the epithelial layer, as far medially as the *plica semilunaris*, trims the free edge of the conjunctiva and leaves a bare strip of sclera, several millimetres in width, between the limbus and the free edge. After extensive experience with this operation over seven or eight years, D'Ombrain states that he has had no recurrences. This is an important claim, for, as D'Ombrain, Gibson and others agree, recurrent pterygium is a major ophthalmic problem. Gibson's experience does not seem to have been so happy. Adoption of D'Ombrain's

[1] *The British Journal of Ophthalmology*, February, 1948.

principles has not meant an end of recurrences, and one wonders whether Queensland climatic conditions are in any way responsible. Gibson analyses various factors associated with recurrence, and some of these will bear further investigation. They do not require comment at the moment. It is important to note that he defines recurrence, an important precaution that, if generally adopted, might eliminate much of the controversy on the subject. How to deal with recurrent pterygium once established is still a problem. Results in Gibson's small series of patients treated with contact X rays are encouraging, and further reports will be awaited with considerable interest. Nonetheless many will share the caution expressed by K. B. Redmond, when the matter was discussed at the Australasian Medical Congress (British Medical Association) last year (see THE MEDICAL JOURNAL OF AUSTRALIA, July 22, 1950, page 159), in emphasizing that the protection of deeper structures is of utmost importance. No doubt the happiest solution, if a trifle Utopian, is the suggestion, made at the congress by J. Bruce Hamilton, of Hobart, that doctors should send their patients with recurrent pterygium to live in Tasmania, where pterygium is a rarity.

CANICOLA FEVER.

LEPTOSPIROSIS is a widely distributed and well-known disease which, like many others, has a surplus of names. Weil's disease is generally understood to be the form of the disease in which the *Leptospira hæmorrhagiæ* is the infective agent, though the name is also used to describe other types. Perhaps it is confusing and unnecessary to use still another and quite different title for the variety of leptospirosis due to the *Leptospira canicola*, but it has a certain value, since it draws attention to the animal vector, the dog. Canicola fever in one respect resembles brucellosis; it is common in canines, just as brucellosis is common in cattle and swine, yet both diseases are relatively rare in man. This is worthy of note in leptospiral infections, for the canine variety has a world-wide distribution, including Australia. Bernard L. Rosenberg reports two cases of canicola fever, and reviews the literature in a recent article.[1] He recalls that this type of the infection was described in 1927, and that the presence of the organism in the kidneys, and therefore in the urine, was then demonstrated. Investigation of the canine strain has shown that it is relatively benign, though this does not prevent it from causing meningeal symptoms, like the other strains. The dog is apparently very susceptible to leptospiræ, although the canicola strain cannot be readily transmitted to the usual laboratory animals. Rosenberg states that the great majority of attacks of canine leptospirosis are due to this strain. The frequency of the infection in the United States of America has been investigated by immunological methods, which showed that of a total of 4800 healthy dogs 27% had demonstrable antibodies for the canicola strain in their blood with a titre of diagnostic degree. This is significant, for, while very high titres are found in the serum of affected dogs, the titres for other strains are comparatively negligible. Tests of dogs brought to veterinarians for various reasons have revealed canicola titres at a diagnostic level in as many as 50% or 60% in series of some 2000 animals. In the past, epidemics of jaundice have been observed in kennels, some associated with a high death rate; it seems certain that the most likely cause of these is canicola fever. It is of interest that two clinical types have been observed in dogs—according to the predominant symptoms, an icteric and an azotæmic type. The former is more often due to the icterohæmorrhagiæ strain, and the latter to the canicola strain. It may also be remarked that these two strains of leptospira cannot be distinguished under the microscope. Turning to the infection in man, we may note that it is curious that man is practically the only animal other than the dog susceptible to the canicola strain. Cases have been described in the human subject since 1934, and Rosenberg finds that the two he now records bring the total in the literature to 200. His own patients made a good recovery, though convalescence was slow in one case. One patient worked for a veterinarian, and the other may have been infected while swimming or from animals. The diagnosis was made by agglutination studies, which showed a very much higher titre for canicola strain than for icterohæmorrhagiæ. The symptoms of canicola fever in man may be briefly summarized. The onset is acute, with high fever and chills; headache is usually severe, conjunctival congestion is conspicuous, and muscular pains are present and associated with considerable tenderness on pressure. Meningism may be observed in the early stages, or after a few days, and gastro-intestinal disturbances are common. The affection of the nervous system includes not only meningeal irritation, but also drowsiness, confusion or delirium. Albuminuria is an early finding, and evidence of mild nephritis is often found; nitrogen retention is usually slight. This is, of course, important because of the known affinity of the leptospira for the kidney, a fact which is significant in the transmission of the disease. Equally important it is to realize that jaundice is not frequent; it is rather stressed as a symptom because of its striking nature. Leucocytosis is usual, and pleocytosis in the cerebro-spinal fluid occurs also in varying degree. Although canicola fever is a considerably milder infection than Weil's disease, the convalescence of both is often prolonged. In the diagnosis agglutination is the most useful method, but when the organisms are found in the urine the finding is convincing. In view of the wide distribution of this disease in dogs, it is possible that mild or subclinical attacks may go unrecognized in man, and that it may be spread more easily than realized by careless handling of animals which may be carriers, or by contact with contaminated water.

[1] *The American Journal of Medicine*, July, 1951.

DRAMAMINE AND POST-OPERATIVE VOMITING.

DRAMAMINE has acquired a considerable reputation for the control of motion sickness, although the validity of some of the reports of its effectiveness has been challenged. On the basis that the mechanism of post-operative vomiting and nausea may be in part similar to that of motion sickness, A. Rubin and H. Metz-Rubin have given dramamine to alternate patients in a series of 250 undergoing operation and state that a statistically significant reduction in post-operative vomiting was recorded.[1] The patients treated received 100 milligrammes of dramamine by mouth forty-five to sixty minutes before the hypodermic administration of the pre-anæsthetic medication, and approximately 80% of them received 100 milligrammes of dramamine orally or rectally after they had recovered from anæsthesia (thirty minutes to two hours). No subject was aware that a special study was in progress. The reduction in post-operative vomiting related to both incidence and severity. The greatest improvement was observed in those patients receiving inhalation anæsthesia, but a decrease in vomiting was evident regardless of the anæsthetic agent employed, the duration of the anæsthesia, the skill of the anæsthetist, or the nature of the operation. Untoward reactions to the dramamine consisted of respiratory depression in seven cases and tachycardia in three; the reactions appeared to be minimal. It was not possible to determine accurately whether dramamine delayed recovery from general anæsthesia, but no striking dissimilarity between the treated and control group was noted in this respect. Side effects were not regarded as serious enough to interfere with the usefulness of the drug. Rubin and Metz-Rubin discuss the possible mode of action of dramamine, but little appears to be known that is definite. However, their clinical experience is of considerable interest and it is to be hoped that others will be able to duplicate their results.

[1] *Surgery, Gynecology and Obstetrics*, April, 1951.

Abstracts from Medical Literature.

RADIOLOGY.

Duodenal Ulcer in Children.

FAY K. ALEXANDER (*Radiology*, June, 1951) states that if the possibility of duodenal ulcer was considered and X-ray studies were carried out more commonly upon children with gastro-intestinal symptoms, it is very likely that a far greater number of lesions would be found and the generally accepted infrequency of the lesion would be disproved. The X-ray diagnosis of duodenal ulcer in children should present no more difficulties than in the adult. If the diagnostic criteria are carefully appraised, autopsy material or surgical inspection to substantiate the diagnosis should not be necessary. Given a patient with a history of generalized or localized abdominal pain, with nausea and vomiting, in whom the symptoms are repeated, the possibility of ulcer should be considered, and appropriate measures should be instituted to establish or exclude the diagnosis. An active duodenal ulcer in children is usually manifested by the presence of an ulcer niche associated with irritability of the duodenal bulb. It is only rarely that one sees the hour-glass, half-bulb, clover-leaf, or pine-tree type of deformity which is so frequently seen in duodenal ulcers in adults. The greater degree of bulbar deformity is usually found in the pre-adolescent patient with symptoms similar to the adult ulcer syndrome of longer duration. The irritability of the duodenal cap and the intermittent pyloric spasm during the early part of the examination are very prominent features. Frequently the bulb is so non-retentive, and throws the barium off so rapidly, that the niche cannot be demonstrated. Not all patients with pronounced pyloric spasm or irritability and non-retentiveness of the bulb will show a niche defect, yet the type of irritability of the bulb is very striking and similar in character to those cases in which a typical niche is seen. Actually, there may be present in such cases a small, shallow, superficial mucosal erosion, not sufficiently deep to collect enough barium to render it demonstrable as a niche on the screen or on the film. In many of these cases, the clinical symptoms, as well as the response to conservative medical regime, are such that a presumptive diagnosis of ulcer is frequently made. It is essential that a niche defect be demonstrated to make the diagnosis of ulcer. Deformity of the bulb with or without irritability is not sufficient. Careful visualization of the pyloro-duodenal region is required, and the ulcer niche will frequently be revealed in the right oblique anterior projection. Eccentricity of the pylorus so commonly associated with ulcer in the adult is usually not seen in children.

The Radiological Aspect of Infectious Mononucleosis.

JULIAN ARENDT (*American Journal of Roentgenology*, December, 1950) states that infectious mononucleosis is a common widespread disease which will frequently come to the radiologist's attention in the form of pharyngeal œdema, virus pneumonia, appendicitis or cholecystitis, leuchæmia or lymphoblastoma, malaria, brucellosis or colitis. Attention is called to the frequent involvement of hilar glands sometimes without or before the development of palpable peripheral lymph glands. Such hilar gland involvement accompanied by considerable splenic enlargement permits the radiologist to suspect the diagnosis, which, however, can be established only by the characteristic blood picture and response to the presumptive and differential antibody test. The lung markings are frequently increased and simulate in every respect the picture of atypical virus pneumonia. These lung changes are due to perivascular and interstitial foci of atypical lymphocytes and in some cases to true intraalveolar infiltration with mononuclear cells. The enlargement of the spleen is evident radiologically and more reliably demonstrated by palpation and percussion of the spleen. The enlargement may persist for months and years after clinical recovery. The frequency of hepatitis with or without jaundice in infectious mononucleosis may lead to X-ray examination of the stomach, in which case hypertrophic gastritis will be found. Examination of the gall-bladder may result in non-visualization of the gall-bladder. An examination of the colon is particularly contraindicated owing to the danger of rupturing the weakened capsule of the spleen.

Malignant Disease in Childhood.

CECIL G. TEALL (*Journal of the Faculty of Radiologists*, July, 1951) states that many of the tumours which in the past have been diagnosed as retroperitoneal sarcomata have now been shown to be neuroblastomata arising in the retroperitoneal ganglia. However, whatever may be the nature of the tumour, there is a simple technique in the radiological examination, which does not appear to be generally recognized, but which may help to differentiate them from kidney tumours. If the direct examination shows what appears to be an enlarged kidney on the side of the tumour, it is always advisable to make a further examination with the patient in the prone position. In the radiograph made at the short distance used in the investigation of the urinary tract the size of the kidney depends on its distance from the film. A normal kidney which is displaced forwards by a tumour behind it will, therefore, look larger than the kidney on the opposite side, but if the patient is turned over into the prone position it will look smaller, for it is now nearer the film.

Influenzal Pneumonia: Recent Experiences.

B. B. HARRISON (*The British Journal of Radiology*, July, 1951) states that pneumonia is the commonest complication of influenza, occurring in all epidemics to a greater or less extent and being responsible for most of the fatalities which occur. There has been a great deal of discussion about whether the influenza virus itself can cause true lung consolidation, but it seems to be accepted now that, whereas in certain cases which are classed as viral pneumonia the causal organism may be the influenza virus alone, the vast majority of the cases of pneumonia occurring in an epidemic are due to secondary invasion of debilitated tissue by other organisms. Without the presence of these secondary invaders, even with symptoms suggestive of pulmonary complications, the chest remains radiologically normal. The pulmonary complications follow a short period of apparent recovery from the acute symptoms of the original influenza. This, of course, corresponds to an invasion of the lung by secondary invaders after resistance has been lowered by the initial virus attack. As far as it was possible to determine, the chest symptoms occurred on an average about seven days after the onset of the original infection, the shortest period being two days and the longest twenty days. Pathologically, the first stage in the involvement of the lung parenchyma is an extension of the initial bronchiolitis into the alveoli. This appears on the radiograph as multiple small areas of infiltration, in the majority of cases concentrated at one or both lung bases. The lesions are larger than miliary tuberculous foci and are soft in appearance with ill-defined edges. Of a series of 62 affected persons examined radiologically 35·5% had this appearance. In these cases the chest was usually clear within five to ten days, and there were no complications. Another 25·8% of the patients in the series had a similar apearance throughout both lung fields, although in one or two cases one side was much more affected than the other. The areas of consolidation vary in size from small areas resembling miliary tubercles to areas with almost the coarseness of metastases. They are, however, always soft with an outline fading into the surrounding lung tissue. It seems probable that these two types of appearances may well be forerunners of the following group of cases. In these there is coalescence of some or all of these sublobular areas of consolidation to give large localized areas of consolidation, neither lobar nor segmental, which may be localized to one part of the chest or be present throughout both lung fields. Patients in this group comprised 20·9% of the series and were by far the most seriously ill as a group. Those patients with isolated patches of consolidation in one or other lung usually returned completely to normal within ten days, but when the consolidation was more widespread, subsequent cavitation sometimes occurred, in spite of intensive antibiotic treatment.

Prolapse of the Prepyloric Mucosa.

PAUL F. J. NEW (*The British Journal of Radiology*, August, 1951) discusses the clinical and radiological aspects of prolapse of the prepyloric mucosa. He states that the diagnosis is essentially radiological. The most important finding is a filling defect in the base of the duodenal cap, immediately above the pylorus. This defect characteristically varies in size and shape during the course of the examination, owing to the relative pliability and softness of the prolapsed folds. The negative shadow, however, is invariably in the base of the bulb, as long as the prolapse persists. The shape and size of the defect depend on the configuration of the prolapsed folds, the degree of prolapse, and the amount of compression applied to the duodenal cap. In some cases the defect is dome-shaped, with a smoothly rounded margin. In others the margin may appear rather square-cut. A very lobulated appearance is uncommon and is probably associated

with polypoid changes in the prolapsed mucosa. The filling defect is usually clearly visible in the barium-filled bulb without compression, but with minor degrees of prolapse, or with partial reduction of larger lesions, the only abnormality seen in the filled cap may be a faint half-shadow at the base, having a crenated margin and extending only a short distance into the bulb. Unless examination is detailed, with graduated compression and the free use of multiple serial films, the appearance may be missed, or its significance may not be appreciated. Manipulation of barium from the antrum into the cap, or spot compression on the prepyloric region, frequently increases the size of the prolapse. In most cases the redundant prepyloric folds can be traced through the wide pylorus to the margin of the bulb defect, the barium streaks in the crevices between the rugæ showing a divergent course. This radial pattern, with the crenated margin, gives an "umbrella" formation. The pyloric canal may appear as a rather broader central band and, when present, is a useful differential diagnostic feature. In a circumferential mucosal prolapse, a secondary mucosal canal is formed, and it is this channel that is seen. In many cases, large, irregular and hypermobile folds are present in the prepyloric region, and are best seen at the greater and lesser curvatures, where they are visualized in cross-section, projecting into the lumen. They may be sufficiently large and reduplicated to produce definite irregular prepyloric filling defects. Antral gastritis and spasm are occasionally associated, as in three cases in this series. More frequently, the antrum appears a little narrowed, with diminished distensibility, but without definite evidence of antritis. The prolapsed folds may slide back into the antrum during the examination, with a decrease in size or complete disappearance of the cap defect and the appearance of a relatively large prepyloric defect.

PHYSICAL THERAPY.

Intravaginal X-Ray Therapy of Malignant Gynæcological Tumours.

R. K. KEPP (*The British Journal of Radiology*, August, 1951) states that in the treatment of gynæcological tumours by radiotherapy, two methods are available—radium and X rays. Radium application by various techniques will mostly remove superficial tumours without difficulty, but the steep dose gradient makes them unsuitable for infiltrations beyond a certain depth. If the amount of radium is increased to give an adequate depth dose to the deeper part of the tumour, an overdose is received by the pelvic organs. Interstitial radium therapy of extensive lesions has not been satisfactory and is followed in many cases by fistulæ, necrosis or hæmorrhage. External irradiation by X rays has the disadvantage that a large volume of healthy tissue must be included. Some improvement can be expected from supervoltage apparatus and perhaps from rotation, pendulum and converging beam techniques. In view of these disadvantages attempts have been made to irradiate, by X rays, malignant infiltrations in the pelvis through the vagina. In contrast to γ rays, X rays are easily screened off from such sensitive organs as bladder and rectum. The author describes an intravaginal hollow anode tube technique, whereby the X-ray beam can be given any required width and direction. Although there is a steep dose gradient, the vaginal wall possesses a high degree of radioresistance, which offsets this disadvantage to a great extent. The apparatus consists of a 60 kilovolt tube, light and easily adjusted. A wide range of applicators has been specially designed, and the choice of the applicator depends upon the shape of the beam and of the tumour to be treated. It is stated that the vaginal wall will tolerate as high a dose as 30,000*r*, the amount required to give a dose, by this technique, of 2400*r* to the pelvic walls. This is usually spread over twelve sessions. In no case is the intravaginal treatment used exclusively; it is always combined with radium or external X radiation. As a rule the radium is placed only in the uterus and no intravaginal radium therapy is used unless the *os uteri* cannot be found. It is claimed that there is no increase in fistula formation, this complication being developed by less than 1% of patients.

Pituitary Irradiation in Prostatic Carcinoma.

WALTER T. MURPHY AND HARRY SCHWIPPERT (*Radiology*, March, 1951) state that the rationale of hypophyseal irradiation for prostatic cancer is based upon the directive functions exercised by the pituitary gland in the entire endocrine system. The effectiveness of orchidectomy and stilbœstrol therapy decreases over varying periods of time, and a new method of altering the chemical environment must be found, which will produce further remission or prolong the benefit from these other procedures. In the cases studied, the dose administered to the pituitary varied from 750*r* in twelve days to 4860*r* in thirty-five days. The authors state that it is difficult to assess the value of treatment, as in most cases orchidectomy was performed in addition to irradiation of the pituitary. However, relief of symptoms occurred in one patient treated by irradiation of the pituitary alone, and in other patients who had suffered relapse after orchidectomy or stilbœstrol therapy. Favourable response occurred in all dosage ranges. It is suggested that depth doses of approximately 1000*r* should be given, to be repeated when relapses occur. A scheme of adrenal irradiation in addition to pituitary irradiation is being investigated.

Thorium X Treatment of Skin Epithelioma, Keratoses, and Delayed Radiation Changes.

J. J. SHER AND WILLIAM E. HOWES (*Radiology*, January, 1951) state that thorium X is a radioactive element with a half-life of 3·64 days. It emits principally α particles with an average penetration in tissue of 0·4 to 0·8 millimetre. Owing to scarcity of radium in Germany during and after the first World War some research was carried out with thorium X, although in most experiments it was used in needles with various filtrations. The authors describe a series of superficial lesions treated by the α radiation alone, thorium X being used in alcoholic solution or incorporated in an ointment. The general method of treatment was to use thorium X in a concentration of 300 microcuries per millilitre of alcohol or per gramme of lanolin. This was applied at weekly intervals up to a limit of eight times. The area to be treated was first cleaned with soap and water, followed by peroxide and finally a grease solvent such as acetone. After drying, the solution was applied with a cotton-tipped applicator to the lesion, and in the case of epithelioma to normal skin for about one centimetre beyond the lesion. The author states that as a rule faint erythema is visible about twenty-four hours after the application of thorium X. This increases for several days and then subsides. The series reported included 51 epitheliomata, all of which had been submitted to biopsy, hyperkeratoses, basal-cell carcinoma and radiodermatitis and radionecrosis. In one case only, that of a deep rodent ulcer, was there failure to respond satisfactorily (over the period of observation). The authors state that thorium X therapy is an efficient method of treating hyperkeratoses and superficial epitheliomata and stimulates the healing in radionecrotic ulcers. The advantages are the ease of application and the absence of sequelæ. The cosmetic result is good and the danger to adjacent parts negligible.

Radiation Therapy of Carcinoma of the Vagina.

FRANK BUSCHKE AND SIMEON T. CANTRIL (*Radiology*, February, 1951) state that primary carcinoma of the vagina is a comparatively rare condition and in general is regarded as having a poor prognosis. Results from the *Institut du radium* and from the authors' series indicate, however, that the prognosis in cases in which adequate radiation therapy is received compares favourably with that of cancer of the cervix. It is essential to differentiate clearly between primary carcinoma of the vagina and carcinoma of the cervix extending on to the vagina. Three stages of the growth are described, according to extent and direction of spread. The figures from the *Institut du radium* show that of 20 patients with growths in stages I and II, 15 remained well. Among the total number of patients in all stages, 15 died, all but two of these within two years. It therefore appears that freedom from local recurrence over three years is a good indication of the final figures. Treatment in nearly every case was by external X irradiation alone or in conjunction with intracavitary radium therapy, and the authors have followed the same plan. Their series consists of ten patients, six of whom have been clinically free of disease for over three years. External irradiation by X rays was given first for all but the very early lesions. Intracavitary radium therapy then followed, the Manchester ovoids being used. This procedure did not follow any routine, but was adjusted according to location and extent of the lesion. In no case, even with massive involvement of the recto-vaginal septum, did a fistula develop. The authors suggest that the generally poor results reported in the literature can be explained by failures of technique, either faulty arrangement of the intracavitary radium or the omission of the external irradiation, which the authors regard as essential.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on June 20, 1951, at the Royal Melbourne Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary medical and surgical staff of the hospital. Part of this report appeared in the issue of November 3, 1951.

Late Syphilitic Infection.

DR. MARGARET HENDERSON presented six cases of late syphilitic infection, with the purpose of illustrating that such cases were still of common occurrence in the out-patient departments, and that treatment and prognosis had considerably changed since the introduction of penicillin.

Dementia Paralytica.

The first patient, a married woman, aged thirty-nine years, had been referred in October, 1949, for the investigation of behaviour disorders, dizziness, diplopia and inability to carry out her household duties. She was wasted and apathetic, with slurred monotonous speech, and unable to answer questions. Psychiatric examination revealed "considerable intellectual deterioration" (Dr. A. J. M. Sinclair). The reflexes were all hyperactive, there were no pupil changes, and slight tremor of the mouth and tongue was present. The blood gave a "+++" reaction to the Wassermann test (14 minimum hæmolytic doses), and the cerebro-spinal fluid also gave a "+++" reaction to the Wassermann test (20 minimum hæmolytic doses). The cerebro-spinal fluid contained 70 milligrammes of protein *per centum* and 10 lymphocytes per cubic millimetre, and yielded a reaction to the colloidal gold test represented by the figures "5554422000" (paretic curve).

The patient was given 10,000,000 units of penicillin, two courses of tryparsamide (18 grammes and 30 grammes), and two courses of "Bismol" (two millilitres per week for ten weeks). Her clinical response was excellent; her general nutrition improved greatly, her mental powers were restored and her social and marital relations returned to normal. Clinical examination now revealed no abnormality. Her serological response was slower. In July, 1950, the blood still yielded a strongly positive response to the Wassermann test. A further lumbar puncture was performed on June 14, 1951; examination of the cerebro-spinal fluid showed that the globulin content was not increased, four lymphocytes were present in every three cubic millimetres, the total protein content was 20 milligrammes *per centum*, and the colloidal gold test produced the following result: "0000000000"; the Wassermann test produced a negative result.

Dr. Henderson pointed out that the use of tryparsamide was now considered by most authorities unjustifiable on the grounds of its toxicity. In several large American series the results of fever therapy *plus* penicillin appeared no better than those of penicillin alone. Although the patient under discussion would almost certainly remain well without further treatment, it had been decided to give her a final course of procaine penicillin (600,000 units daily for fourteen days).

Tabes Dorsalis, Optic Atrophy, Gastric Crises.

Dr. Henderson then showed a male patient, aged fifty-one years, who had been attending the hospital for over six years, and whose serological findings had been negative since 1949. In December, 1949, he had been investigated for severe abdominal pain and diarrhœa. In the absence of other discoverable cause, these were regarded as tabetic. After further iodine and "Bismol" therapy, he was given 8,000,000 units of penicillin, with great relief from the pain and improvement in his gait. On examination of the patient atrophy of the right optic nerve, paresis of the right third nerve, absence of deep reflexes in the left leg and Rombergism were found. He had had some dysuria. No arrest of the optic atrophy had followed penicillin treatment.

Dr. Henderson pointed out that *tabes dorsalis* was notoriously resistant to treatment, and the effects of penicillin were by no means constant. However, the relief of root pains and arrest in the progress of the disease were commonly reported.

Muscular Atrophy Associated with Neurosyphilis.

Dr. Henderson's next patient was a male, aged fifty-seven years. He had bilateral *pes cavus* associated with *spina bifida occulta*, and at the age of three years he had suffered from poliomyelitis, which had affected his left arm and leg. He had been referred to the hospital in April, 1951. After the sudden death of his wife ten months previously he had lost weight and appetite and become weak in the right leg which would not support him. He had some cramps and "rheumaticky pains" in the right leg; he had no parasthesiæ, some blurring of vision and occasional dysuria. On examination of the patient, changes were found in the feet and the left arm and leg associated with *pes cavus* and old poliomyelitis. Ptosis was present, and the pupils were unequal. Pronounced weakness in the pelvic girdle was present, with wasting of the right buttock, and fasciculation in the muscles of the buttocks, thighs and shoulders. The right plantar reflex was extensor. The blood gave a weakly positive reaction to the Wassermann test (four minimum hæmolytic doses). The cerebro-spinal fluid gave a strongly positive reaction to the Wassermann test (seven minimum hæmolytic doses); the protein content was 170 milligrammes *per centum*, the lymphocytes numbered 110 per cubic millimetre, and the colloidal gold curve was represented by the figures "0013410000".

The patient was treated with iodides and with 10,000,000 units of penicillin. At the time of the meeting he was being treated with "Novarsenobillon" and "Bismol". There had been dramatic relief of his pain and improvement in his muscle power. Fasciculation was now difficult to elicit.

Dr. Henderson said that the case probably came within the category of amyotrophic meningomyelitis.

DR. J. GAME commented on the atypical features, particularly the absence of sensory change and the preservation of all deep reflexes.

Aneurysm of the Innominate Artery and Aorta.

Dr. Henderson then showed a married woman, aged fifty-four years, who was known to have had "heart trouble" since 1944, when she was given two courses of "Bismol" injections. The Wassermann reaction was then strongly positive. In 1946 the Wassermann test produced "some fixation", and in 1948 the result was positive only when the test was read in the ice box. At the time of the meeting the result was negative. Further courses of "Bismol" were given in 1947 and 1948. The patient presented a pulsating tumour above the right clavicle, due to an aneurysm of the innominate artery, dilatation of the whole thoracic aorta, and aortic incompetence. There was evidence of obstruction to the right innominate vain and of pressure on the recurrent laryngeal nerve. The pupils were small and irregular, and did not react to light. The ankle jerks were absent. The dorsal segment of the spine was kyphoscoliotic. X-ray examination revealed osteoporosis and some wedging of the vertebræ. There was now some failure of the left side of the heart, for which she had been given digitalis and mercurial diuretics. The gross scoliosis had been attributed to post-menopausal osteoporosis.

DR. G. A. PENINGTON remarked on the intense hyperæsthesia over the dorsal segment of the spine, and suggested that it might be associated with the other manifestations of tabes.

Syphilitic Glossitis and Stomatitis.

The next patient shown by Dr. Henderson was a married woman, aged sixty-seven years, who had had two stillborn children and none living. She had reported in April, 1950, with a history of painful mouth and tongue, of twelve months' duration. There was an atrophic type of glossitis with cracking and scarring of the lips. The Wassermann test produced a "+++" reaction (over 12 minimum hæmolytic doses). She had failed to respond to vitamin supplements and local applications. She was given iodides and mercury for one month, then 7,000,000 units of penicillin, with considerable improvement. Bismuth and arsenic were poorly tolerated, and their administration had not been persisted with. A further 10,000,000 units of penicillin were given in March, 1951, as "Procillin" 300,000 units twice a day. In January, 1951, the Wassermann test produced a positive reaction only when the result was read in the ice box. Leukoplakia persisted for some months and caused some persistence of symptoms. The only abnormality now visible was fine radial scarring about the lips.

Dr. Henderson remarked that as the initial condition had made eating painful, the patient's symptoms were increased by secondary malnutrition. In spite of constant advice her diet was still far from ideal.

Tertiary Syphilis: Polycythæmia.

Dr. Henderson finally showed an unmarried female patient, aged fifty-one years. She had had "enlarged liver" as a child, and aching around the left costal margin since an

attack of pneumonia at the age of twenty-three years. In the last few years she had had frequent migraine attacks and considerable fatigue. Slight nose bleeding and ready bruising were features. In August, 1950, her face and hands presented a congested and cyanosed appearance. The conjunctivæ were injected, and the spleen and liver were both enlarged. Her blood pressure was 200 millimetres of mercury, systolic, and 130 millimetres, diastolic. Examination of her blood showed that the hæmoglobin value was 145% (21·4 grammes per 100 millilitres), the erythrocytes numbered 10,200,000 per cubic millimetre and the leucocytes numbered 8300 per cubic millimetre. Her blood gave a strongly positive reaction to the Wassermann test (six minimum hæmolytic doses); her cerebro-spinal fluid failed to react to the Wassermann test.

She was treated by repeated venesection, iodides and "Bismol". She was admitted to hospital in February, 1951, for a course of penicillin treatment (15,000,000 units). From March 6 to May 22 weekly injections of "Bismol" (two millilitres) and "Novarsenobillon" (0·3 to 0·45 gramme) were given. The last venesection was carried out on March 11.

On May 14 examination of her blood showed that the hæmoglobin value was 93% (13·7 grammes per 100 millilitres), the erythrocytes numbered 5,000,000 per cubic millimetre and the leucocytes numbered 8500 per cubic millimetre. The diagnosis of *polycythæmia vera* was considered unlikely in view of the normal leucocyte count. Antispecific treatment seemed to have effected a reduction in the polycythæmia and diminution in size of the liver and spleen. There was no clinical or radiological evidence of a heart or lung lesion to account for the polycythæmia. Splenic enlargement was rare in acquired disease; the patient's infection might have been congenital.

Discussion.

In a general discussion of the present status of treatment in late syphilis, Dr. Henderson pointed out that penicillin was the most effective spirochæticidal agent known. The course should extend over a period of fifteen days, with a dosage of 500,000 to 1,000,000 units daily. There was a risk of the Jarisch-Herxheimer reaction, particularly in neurosyphilis and aortic disease. As the reaction occurred above a threshold dose, it was not averted by beginning treatment with small doses and increasing them. Most British authorities suggested that bismuth salicylate (0·2 to 0·4 gramme) be given weekly for six weeks before the penicillin, and a further ten weeks' course afterwards. Many American clinics relied entirely on penicillin. The use of arsenic was becoming much less frequent, and tryparsamide was fast disappearing; mercury was quite out of favour. Iodides were useful in the early stages, in the resolution of gummata. Fever therapy had lost its past preeminence in the treatment of parenchymatous neurosyphilis, and its future was in doubt. Aureomycin had activity against the spirochæte, and might be useful in the treatment of the penicillin-resistant or the penicillin-sensitive patient.

Subacute Bacterial Endocarditis.

DR. L. E. ROTHSTADT showed two patients who had suffered from subacute bacterial endocarditis.

The first patient was a female, aged forty-one years, who had been admitted to hospital fourteen months previously. She had had rheumatic fever at the age of seventeen years, and at that time she spent eleven months in bed. After that illness she had felt well until the three weeks before admission to hospital; during that period she had noticed increasing tiredness, loss of weight and loss of appetite. On examination, the patient was a thin, pale woman, with no purpuric spots. Her temperature was 101° F., her pulse rate was 110 per minute and her blood pressure was 130 millimetres of mercury, systolic, and 65 millimetres, diastolic. The heart was slightly enlarged, and systolic and mid-diastolic murmurs were audible at the apex. Crepitations were present at the lung bases and œdema was noted around the ankles and sacral region. The spleen was not palpable. The urine contained albumin, and on microscopic examination a few red blood corpuscles and pus cells and many granular and hyaline casts were seen. Renal function was impaired, and the hæmoglobin value of the blood was six grammes per 100 millilitres. Blood culture produced a growth of *Streptococcus viridans* (penicillin-sensitive) on four occasions. Penicillin, to a total dosage of 2,000,000 units per day over eight weeks, and a transfusion of three pints of packed cells at the commencement of treatment, were given. The response was satisfactory, and she was discharged from hospital after three months. She had felt well since and had resumed her occupation of letter sorter. At the time of the meeting the physical findings in the heart were similar to those on her admission to hospital, but the results of renal function tests had returned to normal. Comment was made on the desirability of recovering the organism from the blood and determining the degree of sensitivity to penicillin, or if necessary to other antibiotics. The necessity of administering adequate doses of penicillin over at least four to six weeks was emphasized.

Dr. Rothstadt's second patient was a male, aged twenty-three years, who had been admitted to hospital eighteen months previously. At the age of three years he had had hæmorrhage from the kidneys, which had been followed by tonsillectomy. During the year before his admission to hospital he had "three attacks of pneumonia", and over the preceding three months he had pain in the left flank, tired easily and lost weight. On examination, the patient was a pale, sick-looking young man. His blood pressure was 135 millimetres of mercury, systolic, and 65 millimetres, diastolic. His pulse rate was 120 per minute. His heart was not enlarged, but a systolic murmur was heard in the second and third left intercostal spaces near the sternum. The spleen was easily palpable. Albuminuria was present, and red blood corpuscles were seen on microscopic examination of the urine. Renal function tests showed considerable impairment. The hæmoglobin value of his blood was 7·5 grammes per 100 millilitres. Soon after his admission to hospital fever was noted, and blood culture yielded a growth of *Streptococcus viridans*. Penicillin was administered to a total of 2,000,000 units per day for eight weeks, and a blood transfusion was given at the start of penicillin treatment. While the patient was in hospital a murmur early in diastole was heard in the second left intercostal space, but it was only after convalescence that the characteristic "machinery murmur" of patent *ductus arteriosus* was audible. He was discharged from hospital after three months, and although he had felt well and had returned to a light occupation, serious renal impairment remained. The question of ligation of the *ductus arteriosus* arose, but because of the renal damage it was decided not to advise surgical treatment.

Cardiac Conditions.

DR. K. GRICE first showed a male patient, aged seventy-one years, who had presented himself to a surgical clinic with the complaint of persistent diarrhœa of several weeks' duration. No cause was found, and he was transferred to the medical wards. He was apathetic and miserable and had a constant low-grade pyrexia. Sigmoidoscopic examination, stool examinations, agglutination tests and blood examination all gave normal results. Eventually blood culture produced a growth of *Streptococcus viridans* on two occasions, and the importance of a mild grade of aortic stenosis, which had been noted on physical examination, assumed greater proportions. Penicillin therapy was begun, his symptoms subsided within a few days, and he remained apyrexial. Several months later a recurrence of similar symptoms began. Though blood culture produced a negative result on this occasion, penicillin therapy caused almost immediate subsidence of symptoms. At no time was the spleen palpable or were petechiæ found. At the time of the meeting, three and a half months since his discharge from hospital, he had remained well apart from recent evidence of mild congestive failure.

Dr. Grice next showed a male patient, aged seventy years, who had been subject to shortness of breath on exertion since his first attendance at the hospital in 1943. Prior to that he had been subject to attacks of rapid, irregular heart action. He had remained under treatment over that period, and in more recent years had had evident peripheral venous congestion and œdema, which responded quickly to bed rest and diuretics. He had a harsh mitral systolic bruit in an enlarged heart with auricular fibrillation. At times a mid-diastolic murmur had been heard at the apex. Fluoroscopic examination revealed an enlarged heart with a fairly straight left border and moderate left auricular enlargement. The electrocardiogram was not diagnostic. He was presented as suffering from rheumatic heart disease with auricular fibrillation and episodes of congestive failure over the past eight years, which responded rapidly to rest and diuretics. There was no certain clinical evidence of tricuspid incompetence.

Dr. Grice then showed an unmarried woman, aged fifty-two years, who had a patent *ductus arteriosus*. The murmur was typical. There had been no alteration in the heart size since she had first been observed in 1946.

General Atherosclerosis.

Dr. Grice finally showed a male patient, aged forty-nine years, who had general atherosclerosis. Cerebral symptoms consisting of tremor of the right hand had begun eighteen months earlier. He had a typical striatal tremor of the right hand and arm and typical facies. Some twelve months prior to the meeting he had become subject to attacks of

paroxysmal nocturnal dyspnœa and dyspnœa on exertion. He had general cardiac enlargement, auricular fibrillation and pulmonary congestion, presumably also of atherosclerotic origin. Six months prior to the meeting he had developed intermittent claudication in both legs. There were no palpable arterial pulses in the right leg or in the left leg below the knee. There was a patch of early gangrene of the right great toe. Dr. Grice said that the patient was presented because of the unusual combination of symptoms of arterial disease in a man of his age.

Allergy.

DR. R. H. O. DONALD, the allergist, and members of the allergy clinic gave demonstrations and showed a number of patients. A cinematographic film on allergy was also shown. Patients were shown by Dr. R. H. O. Donald, Dr. D. Berman and Dr. Gwen Donald. Dr. Hilda Gardner also showed patients, and with the Red Cross workers attached to the allergy clinic gave a demonstration of the various methods of skin testing. Dr. Brian Gandevia showed a number of microscopic sections of pathological processes of allergy in various tissues.

A brief summary of some of the cases is as follows.

A male patient, aged forty-one years, complained of hay fever in the spring and summer for the last seventeen years. He developed hay fever especially when among grasses. He had never had asthma. There was a family history of asthma on the paternal side. Sixteen years previously he had had a course of injections, after which the hay fever cleared up for a number of years. Skin tests by scratch and intradermal methods produced positive reactions to rye grass, cocksfoot, Kentucky blue grass, Yorkshire fog grass, Timothy grass, bent grass, wild oats, cat hair and hen and duck feathers, and a slight reaction to egg white. The patient had symptoms only in the pollinating season, and therefore at present the reactions to cat hair, hen and duck feathers and egg white were being disregarded, and he had been desensitized with an extract of spring grass pollens and had done well during the last season.

A male patient, aged twenty-eight years, complained of hay fever of sixteen years' duration and of asthma of seven years' duration, both of which occurred throughout the year, but were much more severe in the spring and early summer. He had been in a sanatorium with pulmonary tuberculosis for two years, and right thoracoplasty had been performed. He was discharged from the sanatorium on July 29, 1950, with sputum free from tubercle bacilli, and referred to the allergy clinic by the sanatorium authorities. He gave reactions to skin tests with rye grass, Kentucky blue grass, Yorkshire fog grass, cocksfoot, prairie grass, sorrel, wild oats, dock, sunflower, cosmos, dahlia, capeweed, plantain, Iceland poppy, cat hair, dog hair and horse dander, and slight reactions to house dust and flock. He was advised to avoid, as far as possible, the animals to which he was sensitive, and in January, 1951, a course of injections was commenced, the material containing extracts of grass and weed pollens. His condition had greatly improved, and he had had no asthma or hay fever since the end of January.

A female patient, aged fifty-seven years, complained of seasonal hay fever and asthma, of thirty years' duration; she was also dyspnœic. Skin tests produced positive reactions to spring pollens and house dust. She was given three courses of injections of extracts containing spring pollens and house dust, but it had never been possible to reach a very high dosage because of severe local and sometimes general reactions. Nevertheless, her condition had considerably improved.

A female patient, aged seventeen years, had had infantile eczema; this had cleared up at the age of two years. She lived in the same house and had no further trouble until June, 1950, when she developed asthma and perennial hay fever. On being skin tested she gave positive reactions to spring pollens, house dust, cat hair, hen feathers, flock and kapok. She was advised to avoid the substances to which she was sensitive as far as possible. In September, 1950, a course of injections with an extract containing spring pollens and house dust was commenced. She was free of symptoms at the end of 1950, and had remained well since.

A female patient, aged twenty-two years, complained of severe asthma of fourteen years' duration. Recently when she developed a cold she had had a severe attack of *status asthmaticus*. Skin tests produced reactions to house dust, flock, kapok, sheep's wool, horse dander, hen feathers, and dog and cat hair, and slight reactions to spring pollens. She was shown as a patient who was constantly in contact with at least some of the allergens to which she was sensitive, and when there was an added infection such as a cold, bacteria then played a part and the asthma became worse. She was advised to avoid, as far as possible, the substances to which she was sensitive, and she had had three courses of injections of extracts of house dust and mixed influenza vaccine. Her attacks had decreased in number and severity, and she was now to be desensitized by extracts of the other allergens to which she gave reactions.

A female patient, aged fifty-eight years, complained of asthma of eight years' duration; attacks occurred almost every night and lasted for some hours. Skin tests produced reactions to rye grass, cocksfoot grass, Kentucky blue grass, Yorkshire fog grass and house dust. She was given a course of injections of extracts containing house dust and spring grass pollens, and her condition improved greatly. She had one severe attack of asthma in May, 1951, and after that she was retested by the intradermal method, when, as well as giving the positive reactions previously mentioned, she also gave a strong reaction to dog hair and slight reactions to cat hair, horse dander, hen feathers and flock. She stated that she had fondled a dog just prior to the attack in May. She was being desensitized to dog hair.

A female patient, aged fifteen years, worked in a velveteen factory. In January, 1951, she complained of generalized urticaria and itching of over three months' duration. It always occurred when she was working with velveteen. Negative results were obtained to a full range of skin tests by both the scratch and the intradermal methods, but a weal with pseudopodia was obtained with the intradermal injection of histamine (1 in 20,000). She was given a course of injections of histamine azoprotein ("Hapamine", Parke, Davis and Company). Her symptoms subsided in about a month, and although she was still working with velveteen she had had no further attacks.

A male patient, aged fifty-eight years, had been referred by the ear, nose and throat department with Ménière's syndrome. The first attack occurred in 1947, when he had severe vertigo, right-sided headache, deafness and tinnitus. Each attack commenced with nausea and vomiting. The attacks lasted up to one month, and each was severe at first and gradually decreased in severity. He eventually was compelled almost to cease work because of the attacks. All skin tests gave negative results by both the scratch and intradermal methods, except that there was a strongly positive reaction with pseudopodia to the intradermal injection of histamine (1 in 20,000). The patient was being desensitized with histamine azoprotein ("Hapamine"). The treatment had commenced in January, 1951, and the patient had improved greatly and had been able to go back to work. His hearing had improved, but he still had some difficulty in locating the direction of sound, and he had occasional attacks of vertigo; but they were not so frequent or so severe as previously.

Ophthalmological Conditions.

DR. T. A'B. TRAVERS, DR. N. LEWIS, DR. J. BRYAN FOSTER and DR. F. WILLIAMS showed patients with eye disorders. There were several patients who had common fundus conditions. A patient with tuberculous keratitis treated by a course of streptomycin and para-aminosalicylic acid was slowly improving. The para-aminosalicylic acid was still being given. Several patients with corneal grafts were shown; one patient had had both eyes grafted successfully, the earlier graft having been carried out two years previously.

Painful Shoulder.

Periarthritis.

DR. LEIGH WEDLICK first showed a male patient, aged fifty-nine years, who had had anginal attacks for two years.

On March 3, 1950, he had been referred with pain and stiffness in the right shoulder since a fall six months earlier. X-ray examination revealed no abnormality. Moderate limitation of all shoulder movements was present. The condition was considered to be periarthritis. Short-wave therapy, massage and exercises were given two or three times weekly. Regular records showed a steady increase in the range of movement. By June 6 the pain had almost gone. There was still slight limitation of shoulder movement, but the range of abduction had increased very considerably. The patient was discharged from hospital to carry on exercises at home. On September 8 he reported that he was free of pain and had a full range of movement.

Tendinitis with Calcification.

Dr. Wedlick then showed a female patient, aged forty-eight years, who on February 8, 1951, reported that for six weeks she had had pain in the left shoulder radiating to the forearm, more severe on movement and with a "catch"

pain. There was moderate limitation of abduction and slight limitation of internal rotation. Dawborn's sign was present. Tenderness was elicited over the bicipital groove. X-ray examination revealed calcification in the supraspinatus tendon in both shoulders, slight in the right, pronounced in the left. Short-wave therapy and exercises were given twice a week. By April 3 the patient had a full range of movement and the pain had gone. Dr. Wedlick said that the presence of symptomless calcification, as in the right shoulder, was not uncommon.

Osteoarthritis at the Glenoid.

Dr. Wedlick's third patient was a female, aged sixty-seven years, who had presented herself on April 13, 1951, with a two years' history of progressive stiffness of the right shoulder, with pain radiating down the arm to the hand. Shoulder movements were greatly limited. X-ray examination revealed gross osteoarthritis at the glenoid, with almost complete loss of cartilage and pronounced lipping. Diathermy, massage and exercise were given three times a week. By June 12 the patient had had great relief of pain and considerable increase of movement; the range of abduction had increased from 75° to 105°, that of flexion from 72° to 60°. It was remarked that osteoarthritis at the glenoid was uncommon and rarely seen to the gross extent shown in the case under discussion. Moreover, it was usually rather refractory to treatment.

Osteoarthritis of the Knee.

Dr. Wedlick's next patient was a male, aged eighty-five years; for five weeks he had had pain and stiffness in the left knee. He had been given lactic acid and procaine injections into the joint without relief. On February 5, 1951, some stiffness was present, with grating in the left knee and weakness of the quadriceps. X-ray examination revealed severe osteoarthritis. Short-wave therapy and resistance exercises were given three times a week. By April 9 the patient was free of all symptoms. On June 20 he reported that he was well. Dr. Wedlick remarked that in the treatment of osteoarthritis of the knee with physical measures, the average duration was seven weeks, and in 85% of cases complete or considerable relief of symptoms was achieved.

Osteoarthritis of the Hip.

Dr. Wedlick next showed a female patient, aged fifty-two years, who on February 16, 1951, had given a history of pain in the right hip radiating to the thigh and knee, of fifteen months' duration; the pain was causing a pronounced limp. Flexion and rotation of the hip were limited, and early flexion deformity was present. X-ray examination revealed gross osteoarthritis of the right hip. Treatment, given three times a week, consisted of short-wave therapy, massage, exercises, and correction of flexion deformity. On May 4, after 23 treatments, considerable but not complete relief of pain had been achieved; the limp had gone, and the flexion deformity had been corrected. The range of movement of the joint had been increased.

Sciatica.

Sciatica Secondary to Gluteal Fibrositis.

The next patient shown by Dr. Wedlick was a male, aged fifty-seven years, who on March 13, 1951, complained that he had had sciatica on the right side, radiating down the outer aspect of the leg and the foot, for twelve months. X-ray examination revealed no abnormality. Tenderness was present in the right gluteus muscle, Lasègue's sign was absent and the ankle jerks were present. The spine and hip movements were good. Twenty millilitres of "Novocain" were injected into the right gluteus muscle with immediate relief, which partially relapsed next day. Short-wave therapy, massage and exercises were given, and on April 24 the patient was free of pain and working as a labourer.

Diskogenic Sciatica.

Dr. Wedlick then showed a male patient, aged twenty-five years, who had strained his back seven months prior to his presenting himself at the hospital on March 13, 1951. Since then he had had sciatica on the left side, radiating to the outer aspect of the leg and the foot. Lasègue's sign was absent on the right side and present on the left side (at 20°). There was no local tenderness. The left ankle jerk was diminished, spine flexion was limited, and X-ray examination revealed a narrowed disk space. The patient was treated by manipulation of the back without general anæsthesia; it was followed by short-wave therapy, massage and exercises three times a week. By April 18 the pain had gone, and Lasègue's sign was absent; spine flexion was almost normal, and the patient could reach to within two inches of the floor with straight knees. On June 20 he reported that he was well.

Sciatica Secondary to Spondylitis.

Dr. Wedlick next showed a female patient, aged sixty-one years, who had presented herself on March 16, 1951, complaining of pain in the left hip of two years' duration; the pain radiated down the thigh and knee, and had become much more severe in the last six months. On examination of the patient, it was seen that her spine was stiff and that she adopted the "head forward" posture. Lasègue's sign was absent. X-ray examination revealed osteoarthritis of the lumbar part of the spine and the sacro-iliac joints. By April 18 she was free of pain, and on June 20 she was reported to be well.

Painful Amputation Stump: Percussion Technique.

Dr. Wedlick finally showed a male patient, aged nineteen years, who had presented himself on December 19, 1950. Twelve months previously amputation of his right great toe had been necessary as the result of an injury. He still had a limp, and the stump was acutely tender and painful. "Novocain" was injected and ten minutes' hard percussion was carried out, in accordance with the method of treatment described by Russell and Spalding (*British Medical Journal*, July 8, 1950). The patient had been completely free of pain and limp ever since.

Paraffin Wax Baths in the Treatment of Stiff Fingers.

A demonstration was also given of the use of paraffin wax baths in the treatment of stiff fingers due to rheumatoid arthritis or resulting from trauma.

Indolent Leg Ulcers Treated by the Bisgaard Method.

DR. HOWARD HALPER showed four patients with indolent leg ulcers treated by the Bisgaard method. Three of the ulcers were of varicose origin and one was trophic. Two of the varicose ulcers, both of which were of long standing, had healed completely within two months. The third, which was an ulcer of some twenty years' duration, was healing satisfactorily after six weeks' treatment. The trophic ulcer, which had arisen after a Britens arthrodesis, had begun to heal satisfactorily after the institution of the Bisgaard method. An interesting feature of the treatment was the relief of the varicose eczema as a result of the treatment.

Torula Granuloma of the Lung.

DR. IAN MCCONCHIE presented three patients who had had torula granuloma of the lung treated by resection.

The first patient, a married woman, aged thirty-eight years, had had torula meningitis, a torula granuloma in the subapical segment of the upper lobe of the left lung, and an extrapleural torula granuloma adjacent to the neck of the left fifth rib. For three months prior to her admission to hospital in December, 1950, she had suffered from severe headaches; blood-stained sputum appeared three weeks before her admission and persisted. Pre-operative investigations revealed torula in the cerebro-spinal fluid, torula in the sputum, and an opacity in the subapical segment of the upper lobe of the left lung. Dr. Graeme Robertson and Dr. J. Hayward decided that the lung lesion should be resected in spite of the presence of proven torula meningitis. In January, 1951, Dr. Hayward resected the subapical segment of the upper lobe of the left lung and removed an extrapleural granuloma from in front of the neck of the left fifth rib close to the termination of the intercostal vein. Post-operatively the patient developed a small apical empyema, which healed rapidly. After operation her severe headaches disappeared, torula disappeared from her cerebro-spinal fluid, although some increase in cell content persisted, and torula disappeared from her sputum. Six months after operation she was free of headache, she had no sputum and the X-ray appearance of her chest was quite normal. On section the lung lesions were found to be teeming with torula organisms. Dr. McConchie commented that there were two points of interest in the case under discussion. The first was the proximity of the lung and extrapleural granulomata to the point where the intercostal veins joined the spinal venous plexus; it was possible that infection spread from the lung to the meninges via the spinal venous plexus. The second was that pulmonary resection was carried out in spite of the presence of meningitis, with the idea of preventing reinfection of the meninges.

Dr. McConchie's second patient, a man, aged twenty-eight years, employed in a biological laboratory for four years, had had a large torula granuloma in the lower lobe of the

left lung, the diagnosis being proved pre-operatively by aspiration biopsy. In 1942 he was found to have bilateral pulmonary tuberculosis with organisms in the sputum. Subsequently an artificial pneumothorax was induced, and this had obliterated itself by 1945. He then returned to work, symptom-free, and with a radiologically normal lung field. He remained well and his lungs were clear radiologically until April, 1949. At that time he felt as if he had influenza, lost weight, and developed a cough with blood-stained sputum. X-ray examination showed a large spherical mass in the lower lobe of the left lung and a small spherical mass in the upper lobe of the right lung. It was thought that these were probably tuberculomata, and very full investigations to exclude other causes of the masses yielded negative results. He was given streptomycin, and the lesions decreased to half their original size and the quantity of sputum diminished. However, during the next two months no further decrease in size occurred, and the possibility of neoplasm or chronic abscess was seriously considered. Aspiration biopsy of the large mass in the lower lobe of the left lung was performed, and torula was demonstrated in the material obtained. In August, 1940, Dr. Hayward performed a left lower lobectomy, and a granuloma, again teeming with torula, was demonstrated in the resected lobe. The post-operative course was entirely uneventful. Dr. McConchie said that since operation the patient had developed no symptoms suggestive of meningitis, he had remained free of sputum, and the upper lobe of his left lung had remained normal. The small lesion in the upper lobe of the right lung had progressively decreased in size after operation until March, 1951. However, there had been a slight increase in the size of that lesion between March and June, 1951, and the possibility remained that resection of the right-sided lesion might have to be performed.

Dr. McConchie's third patient, a man, aged forty-two years, was an example of a common problem—the patient with a circumscribed opacity in the chest X-ray film, undiagnosed pre-operatively in spite of full investigation. The patient presented was symptom-free and his chest X-ray film a year previously had been quite normal. A routine X-ray examination, made one month before his admission to hospital in October, 1949, showed a spherical opacity, one inch in diameter, in the lower lobe of the left lung. Full investigation failed to reveal the cause of the shadow. It was decided that thoracotomy should be performed, and in November, 1949, Dr. McConchie performed a left lower lobectomy. The lobe contained a granuloma, again teeming with torula organisms. The patient's post-operative course was uneventful. Since operation he had developed no symptoms suggestive of meningitis, he had remained sputum-free and his chest X-ray films had been quite normal.

Dr. McConchie said that the three patients formed an interesting small series: the first, with a lung granuloma and meningitis, diagnosed pre-operatively by recovery of torula from the sputum and the cerebro-spinal fluid; the second, with a past history of pulmonary tuberculosis, diagnosed pre-operatively by aspiration biopsy of a large granulomatous mass; the third, with the circumscribed opacity in the chest X-ray film, undiagnosable except by thoracotomy and resection.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

TO THE EDITOR OF THE SYDNEY GAZETTE.

[*Sydney Gazette*, April 20, 1827.][1]

Sir,

An individual here named Elder, a baker, having endeavoured to excite the minds of the people in this neighbourhood against me, on account of having anatomically examined the bodies of several persons who died, and the public press having been stated as the medium through which such is intended, I deem it a duty incumbent on both myself and the profession to which I have the honour to belong to state to you the facts of the case which I am ready to verify on oath, and I consider it the more necessary because the Public of Great Britain have had their minds feverishly excited by such means, the consequence of which is, that medical students are obliged to go to Paris for dissection and study and with such cramped measures, the race of surgeons who are now springing up, will be less competent to decide on the safety of the lives of their fellow creatures. A woman named BORE, having died some time ago, I received the consent of the people who were surrounding her, to examine her body which I conceive is the duty of every surgeon, if he has any regard for the welfare of the living. I did investigate the ravages which the drinking of ardent spirits had produced, and a long tirade was given to Mr Wentworth who spoke about it at the Supreme Court.

The woman named JANE BORE, left some property at her demise, which fell into the hands of a Government man named HOPKINS with whom she had been cohabiting and among other bills there was a certain promissory note, drawn by JAMES ELDER in her favour, in payment for a beautiful house where MAJOR LOCKYER'S family reside. This house was built by Jane Bore's husband, but he having another wife in England went home to her, and sold this cottage to Mr Elder for £60 sterling payable in twelve months to Jane Bore his wife. After her decease I applied to Mr Elder to be paid for my trouble as he had this £60 still in his possession. Elder said he had given £1 to Mr WENTWORTH for advice and that Mr W had told him never to pay a fraction of it. I brought the matter before the Supreme Court and although Dr. Harris had cautioned the prisoner HOPKINS not to part with this note yet Mr Elder bought the note for £60 from him by giving him £20, having commissioned HUGH TAYLOR to get it for less if he could: this was sworn to before the Bench of Magistrates who censured Mr Elder's proceeding. On the trial coming on before the Supreme Court the Chief Justice took an exception to the clause in the Act of Parliament, as not applicable in this colony. Since this period Mr Elder has been stirring up the people to prevent anatomical investigation and a woman having died here, under very suspicious circumstances, I examined minutely the body and contents of the stomach, fearing that poison had been given, having the Coroner's sanction for so doing. Yet a petition was presented to the Governor praying an examination into my conduct for mangling the body as was stated. The case which called forth these remarks is as follows:—A man named Peter Kinshela (free) a bricklayer in the employment of Messrs LENTZ and BATMAN, was placed under my care nine days ago, labouring under a severe illness, for which he had consulted DR BLAND some time ago: he was much reduced in appearance, and after calling on me twice, he was unable to walk a short distance to consult me, and I attended him at the house of his employers LENTZ and BATMAN. I visited him 2, 3 and 4 times a day until yesterday when he died. As this man had been under my care in a hospital in Dublin 7 years ago and as he had now died from the diseases produced by drinking of spirituous liquors to which he had been much addicted I applied to Messrs Lentz and Batman for permission to examine his stomach: they both said they had no objection and they would consult a friend or two of his. I also applied to the Roman Catholic clergyman who told me he had no objection and I had better use my own discretion. I applied to his friends who gave permission and were present when I commenced and finished.

I found on examination, extensive disease of the stomach liver and spleen and the lungs full of sabulous particles and tuberculated and, as is generally the case, the body was much emaciated, while the liver and spleen had increased to an enormous size. I made as little cutting and sewing up as possible and having shewn to a number of bystanders the effects produced by drunkenness I endeavoured to impress on their minds the impropriety and punishment of such a sin.

This morning (Wednesday) the Coroner called on me to say, that an Inquest was to be held on PETER KINSHELA'S body, a rumour having been raised that Messrs LENTZ and BATMAN had neglected him and requesting my attendance: at this inquest my having opened the body became the subject of high and inflammatory discussion by Mr Elder, and he having stated that the medicine I gave him caused his death, I called in the evidence of Surgeon DALHUNTY and I demanded a re-examination of the body before Surgeon D: he did examine anatomically the body of the deceased and gave his opinion as follows:—That he perfectly coincided in my opinion, and that the deceased came to his death in consequence of excessive intoxication: that under any circumstances he could not have lived more than a few days, that there was a perfect disorganization of every viscus, and that I had made as little disagreeable marks as it was possible to have done.

[1] From the original in the Mitchell Library, Sydney.

The Coroner's Jury having sat for some time, a verdict was brought in by the Foreman and nine Jurymen that the deceased died by the Visitation of God, while three Jurymen, viz. Mr. Elder and two friends of his would not give any opinion because the body had been opened before they saw it—Mr Elder strongly insisting that a remonstrance should be sent to the Governor to prevent my opening any body. Now, Mr Editor, the necessity of morbid investigation is a circumstance of which every enlightened man of the present day coincides and I sincerely hope that, through the medium of Mr Elder, no outcry will be raised against the examination of dead bodies, which bodies can be of no other utility, but that of affording instruction to the living. The Coroner, Foreman and nine Jurymen, have expressed themselves highly satisfied with my conduct, as there was not the slightest reason for me to suppose that an Inquest would be necessary, and consequently that I did not act prematurely, and if thought requisite, they are ready to testify to this statement, should it be brought before public view. Hoping that the minds of the Inhabitants here will not be enflamed and excited against such anatomical investigations. I have the honour etc.

ROBERT MONT MARTIN,
Surgeon.

Correspondence.

PRE-BRONCHIECTASIS.

SIR: In reference to my recent paper on pre-bronchiectasis two letters have appeared in the journal—by Dr. Monk on September 22 and by Dr. D'Arcy Sutherland on October 6.

The main complaint of both these writers seems to be against bronchoscopy. In fact Dr. Monk says that many patients have done well without "ever having been subjected to bronchoscopy". That expression would make it appear that bronchoscopy is something to be dreaded. I will admit that at times this could be so, as pointed out by Dr. Blashki in the discussion on my paper. However, if efficiently done by one who is expert in the use of the bronchoscope there is nothing very disturbing about the procedure. If it was so uncomfortable, I am sure we would not have, as we have had, quite a number of little children, down to four years of age, asking their mothers if they can come up for extra bronchoscopic treatment under local anæsthesia. These children as well as adults recognize the benefit they get from the treatment.

These writers stress the coughing of the patient during a bronchoscopy. A coughing patient is uncommon in my clinic unless they should cough under instruction. If the local anæsthesia is efficiently applied and the bronchoscope correctly used, these disturbing factors should not arise. As with all surgical procedures, the dexterity necessary can only be acquired by long experience with many cases.

I have had patients from all chest, thoracic and bronchoscopic clinics in New South Wales come to my clinic and stay there for treatment, expressing their approval of the treatment as compared with what they had previously had.

I have found these patients respond well to bronchoscopic lavage, and I will look forward with keen interest to early publications by these gentlemen of a series of cases with a full follow-up as treated in their clinics.

Yours, etc.,

229 Macquarie Street, H. B. HARWOOD.
Sydney,
October 26, 1951.

SIR: Mr. Sutherland's letter (THE MEDICAL JOURNAL OF AUSTRALIA, October 6, 1951) prompts me to add my own comments on Dr. Harwood's article. They were that the treatment advocated is economically impracticable and in most cases quite unnecessary. I know that bronchoscopy

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 20, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.[2]	Australia.[3]
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	4(3)	..	..	..	1	..	5
Diphtheria	4(3)	2(1)	1	3(3)	2(2)	..	..	..	12
Dysentery (Bacillary)	..	3(1)	3(3)	..	..	..	..	..	6
Encephalitis	..	2	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	..	..	..	12(8)	..	..	..	12
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	5	..	..	..	..	..	5
Malaria	..	..	1(1)	..	..	..	1	..	2
Meningococcal Infection	1	3(2)	..	1(1)	3(2)	..	..	..	8
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(4)	5	2(1)	33(24)	2(1)	..	..	..	50
Puerperal Fever	..	..	1	..	1	..	..	..	2
Rubella	..	14(2)	2	..	3(2)	..	..	..	19
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	24(14)	16(10)	3(3)	7(6)	2(2)	..	..	..	52
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1(1)	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	40(33)	12(8)	16(8)	7(4)	18(13)	7	..	..	100
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Australian Capital Territory

by experts is considered a quite safe procedure, but mishaps do occur, I believe, and I also believe that the pre-bronchiectatic state can be removed by simpler and certainly more comfortable methods.

The first essential is that parents shall realize that malaise or cough persisting after measles, pertussis or "bronchitis" are serious matters to be taken to the doctor; the second that the family doctor shall remember the possibility of pre-bronchiectasis and insist on early routine X ray in such cases. This will show, rarely, the localized segmental collapse, which is solvable by bronchoscopy and suction, or, more commonly, the multiple mottlings of residual broncho-pneumonia, which are, I think, the common precursors of the generalized bronchiectasis of the lower parts of the lungs, which is the common type of case.

The indications here are for the reestablishment of patency, the reexpansion of collapsed alveoli and control of infection. Mr. Sutherland advises postural drainage, steam inhalations and breathing exercises. My own routine is to add penicillin aerosol by "Carbogen" and short-wave therapy to these things—or rather to rely on them as principals and use the others as adjuvants. "Carbogen" is an irresistible agent for producing full respiratory movement, aerosol takes the antibiotic to the spot where it is needed. Short-wave diathermy may be of dubious value, but it used to work in the slowly resolving pneumonias of the pre-antibiotic era, so I keep my faith in it.

Small children may or may not cooperate in breathing exercises. But the strongest of voluntary movement may be inhibited by reflex from inflamed tissues. The primitive wide respiratory excursion of carbonic acid excess will, I think, brook no inhibition. That is why I think it better than the coaxings of the physiotherapist. The continuance of respiratory education after the chest is clear and until proper breathing habits are established is, I think, important as a preventive of recurrence. But its place in the active treatment of the condition is secondary to "Carbogen".

These things are, after all, an expansion of Mr. Sutherland's treatment, and I agree with his thesis. They will clear up most residual bronchopneumonias in which fibrosis has not started in a week or two. There will be a small residue in which Mr. Harwood's treatment may be necessary and of avail. But its general use savours of shooting at sparrows with heavy artillery.

Yours, etc.,

163 North Terrace, Adelaide,
October 22, 1951.

CYRIL T. PIPER.

Obituary.

WILLIAM CHARLES TEESDALE CHAMBERS.

WE regret to announce the death of Dr. William Charles Teesdale Chambers, which occurred on October 29, 1951, at Adelaide, South Australia.

The Royal Australasian College of Physicians.

THE following were admitted to membership of The Royal Australasian College of Physicians at its recent meeting in October, 1951: Dr. B. T. Dowd, Dr. H. M. Landecker and Dr. A. A. Young, of New South Wales; Dr. R. T. J. Galbally, Dr. T. H. Hurley and Dr. A. W. Venables, of Victoria; Dr. R. B. Lefroy, of Western Australia; Dr. A. E. Erenstrom, Dr. C. H. Garlick and Dr. T. H. Pullar, of New Zealand.

Corrigendum.

IN the article by J. C. Edwards entitled "The Osmotic Theory of Eclampsia and the Mechanism of Water Retention", published in the issue of October 27, 1951, at page 559, the legends under Figures IV and V have been transposed. The legend appearing under Figure IV applies to Figure V and vice versa.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Duggan, John Malcolm, M.B., B.S., 1951 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Kinny, Noel Wesley, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Diary for the Month.

Nov. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 21.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 22.—Victorian Branch, B.M.A.: Executive Committee.
Nov. 23.—Queensland Branch, B.M.A.: Council Meeting.
Nov. 27.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 28.—Victorian Branch, B.M.A.: Council Meeting.
Nov. 29.—New South Wales Branch, B.M.A.: Branch Meeting.
Nov. 29.—South Australian Branch, B.M.A.: Branch Meeting.
Dec. 4.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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